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FOREWORD

ALBERT B. FERGUSON, JR., M.D.

From the practical viewpoint there is no way of compressing the subject material that most commonly binds orthopedics and pediatrics together into a single volume such as is here presented. The subjects selected are those which would be most likely to elicit a high reader interest because they vex one daily and yet contain information not generally known, so that their discussion will aid the practitioner faced with diagnosis and disposition. No attempt has been made to select either highly technical or surgical subjects.

Knock knees, bowlegs, pronated feet and poor posture confront us all every day. We are always at the borderline between the normal and the abnormal. Growth helps overcome many deformities, but equally can turn what we thought was a minor fault into a serious cosmetic and functional calamity. In the present temper of the people of this country not only are preventive measures encouraged, but also the failure to carry them out is regarded as a serious fault by the parents of the otherwise well child. The material success of this country supports measures that in other regions of the globe would be regarded as luxuries by the practitioner so busy that he cannot begin to cope with the severe, last-stage deformities confronting him. We are fortunate to be able to give attention in such detail in trying to achieve an ideal in which orthopedics and pediatrics are aligned: a person who is able to use talent and ability to the fullest without hindrance from poor health or deformity.

The examination of the school child, the recognition of some portion of the posture problem, some unanimity of thought regarding pronated feet and shoes may help toward this end. The care of poliomyelitis and tuberculosis, when properly done, reaps big dividends; when neglected, leaves enormous reconstruction problems to those who eventually gather them in.

Growth inequality and the possibilities for overcoming its effects should interest even those who seldom come across this disturbance.

The childhood limp is a practical and sometimes vexatious subject. There is always an underlying cause, and the mere knowledge of some possibilities speeds their recognition. Synovitis of the hip is so common among these and surprisingly little known as an entity.

When we wander out into the land bordering between normal and abnormal, it is well to remember that not everything is good merely because it exists as an anatomic structure. Our legs must be good mechanical structures to bear up under the punishment of a lifetime of weight bearing. Growth itself may be a cause of disease.

All the subjects presented here were chosen because it was felt that the authors of these topics could supply information of value in the solution of the day-to-day challenge which these probems present. None of the more complicated subjects, such as cerebral palsy, reconstruction of the hand, slipped epiphysis, coxa plana, and none of the technical subjects that make up Orthopedics are included. The reason is that this is a volume *to aid in the office practice of pediatrics as related to orthopedics* rather than a text of orthopedic surgery as related to pediatric subjects.

ORTHOPEDIC EXAMINATION OF THE GROWING CHILD

CHARLES WEER GOFF, M.D.

In most areas within these United States a boy or girl is considered legally a child until 18 years of age or, in some areas, as old as 21 years.[8] With our modern sophistication and relative precociousness of youth in cultural acquisitions, perhaps the former is, or will be at any rate, a more acceptable age limitation. For practical purposes most pediatricians consider age 12 as the point of departure when a child becomes relatively an adult. In this discussion the child, whether male or female, will be considered full blown at age 16, and from that age onward should be rated an adult. In the physiologic sense this is reasonable. Both sexes have reached the reproductive age, which is the natural "Rubicon" dividing childhood from adulthood.

SCOPE

Those who deal with children from day to day inevitably accumulate a fund of impressions and significant ideas, enabling them to quickly appraise disorders, to rapidly screen the normal from the abnormal and to formalize their interpretations. The orthopedist, in this regard, is concerned with the whole child. Usually a comprehensive examination is called for, not only to detect a lurking disability, but also to establish a number of base-line determinations, relative to the child's physical growth, body type, individual metabolic activity, ossification age, pubescence, symmetry, gait, coordination (nerve-muscle control) and posture.

The orthopedist,[9] together with pediatricians and many co-workers, recognizes present day growth concepts as tentative, with no formulated "laws," so comforting when known; yet there are many easily

followed "signs," to use a western expression. An orthopedic examination consists in "reading these signs." We know that a child grows. The dynamics or nature of his or her growth can be measured. These time bombs of growth will be discussed, together with their fluctuations, their correlations as to sex and orthopedic disorders, as influenced by heredity, environment, trauma and the endocrines.

EXAMINATION SET-UP

An infant must be completely disrobed. All children should undress to at least their underclothes and socks. Some physicians use a light examining gown; this is necessary for the 10 year old girls and older. A sense of privacy is especially keen at this age and should be respected.

A long room, relatively narrow, is preferred. At one end is a large mirror, and on the floor is drawn a white line. As in Figure 1, the ex-

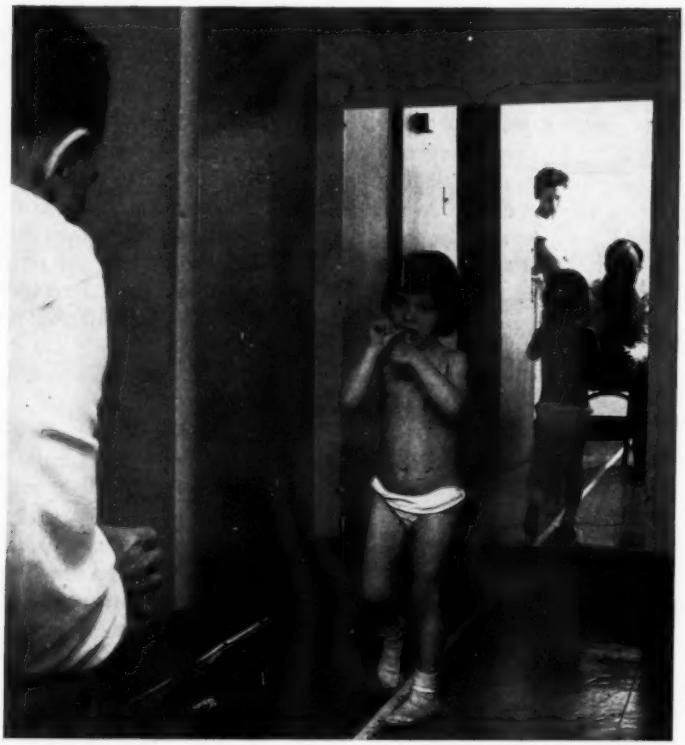


Fig. 1. Examining room setup. The white line, the mirror and the position of the examiner are designed to give full opportunity to observe effectively.

aminer sits at one end and is able to look along the line and observe the child in both planes, either directly or in the mirror, or both simultaneously. This arrangement brings out posture, gait, symmetry, body mechanics and other desirable dynamic aspects in a most satisfactory manner.

The examining table should be large enough to use for a mature person, covered and flat, with at least two pillows for adjunct positioning

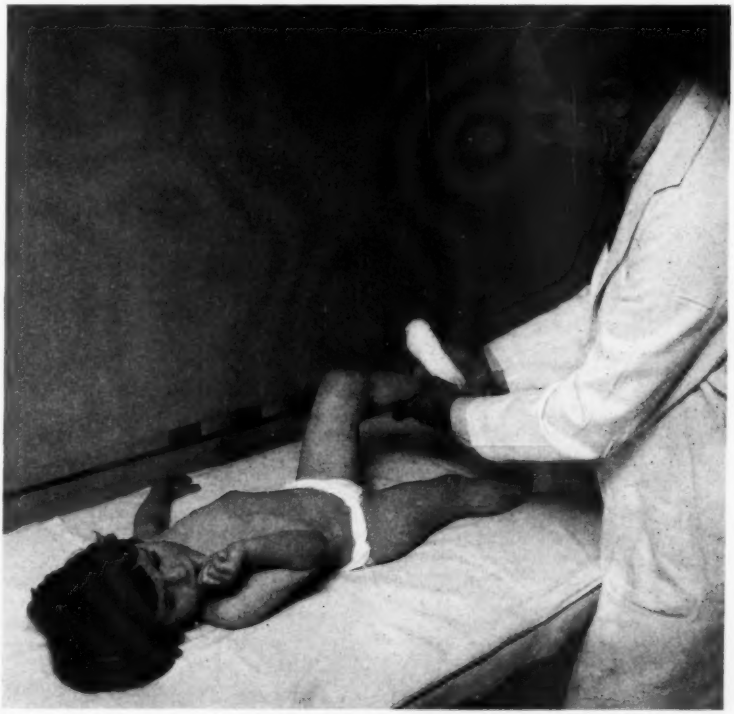


Fig. 2. Examining table. This large size permits easy access and freedom of movement during examination.

as in test exercise 4 (Fig. 16). All joints are tested for full ranges of movements. Tissue tone is palpated, together with the usual testing of coordination, reflexes, strength of muscles in their various systems and any other aspect consistent with a desire to come to a complete awareness of the full or total child. This must be emphasized.

Infants

The infants' posture is noted,[10] e.g., horizontal in type, back without

a lumbar lordosis, extremities equal, kicking and responsive to reflex stimulation. Unequal extremities, flaccidity, marked external rotation, talipes of one type or another, are all apparent if present. The symmetery of normal is a great aid. Is the cranium enlarged? Are the heel cords slack? Are there any accessory appendages, toes? These are noted at a glance.

At six months or more of age, back and abdominal muscles should be strong to permit sitting. If not, the infant slumps forward. Potbellies are common, probably because of the high liquid component in diet of that age group.

When an infant becomes one year old or more, the walking gait is added to the test. Are the knees abducted? Are the feet toeing in or out? The early normal gait is one with lower extremities spread out, uppers in the air, often over the head for balance. A wide range of normal must be appreciated at this time.

Year Olders

After the child has learned to walk the wide stance slowly shifts to one with the knees closer together. At first the child may show considerable distance between its knees when its ankles are held together, or a considerable distance between its ankles when its knees are held together. Knock knees or bowlegs at this age are within normal ranges. Nothing should be done about them unless the degree exceeds normal limits.

Two and Three Years Old[9]

As the child grows into the two- and three-year old group, the knock knees or bowlegs of infancy may persist. Often when the knees are together, the ankles are 1½ to 2 inches apart. This knock knee or bowleg stance is indicative, in part, of the child's somatotype or body build which will persist throughout the rest of life. The fat type of child is likely to show knock knees, with an occasional youngster showing straight legs or legs slightly bowed. This latter child is apt to be a muscular type.

Standards are used for rating knock knees, bowlegs, pronated feet, hyperextended knees, back knees and slumped back, as well as lordosis in the lumbosacral region and protruding abdomen. A child can be rated at six months' intervals against these standards.

The child's somatotype or body build is a genetically conditioned continuum.[9] This can be determined by computations. The body type can also be rated from photographs taken according to the Sheldon-

Hooton technique. This is admittedly difficult, but lends itself to an inspectional appraisal. The body build will influence the child's entire physical and mental make-up throughout the rest of its life. A study of the child's parents is, of course, of tremendous value at this time. If the

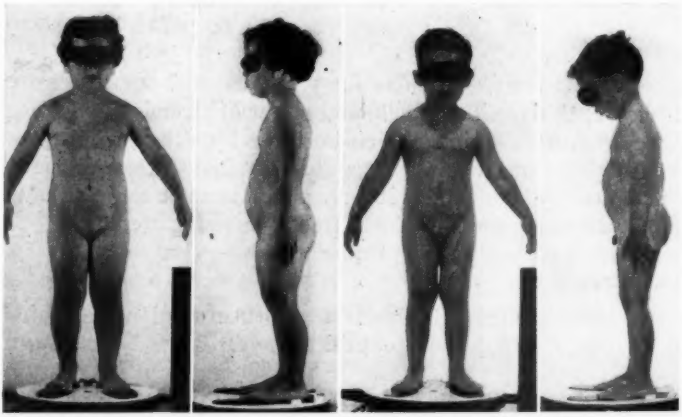


Fig. 3. Girl, age 7 years, weight 50 pounds, stature 42.3 inches; boy, age 4 years, weight 45 pounds, stature 41.5 inches; indicating robust health with the marked sex differential frequently observed.

father and mother are both of the fat type, it will be outside the laws of probability for the child to be of another type.

Middle Childhood

From the work that has been reported before, and the abstracts of material from other studies, it is apparent that the five, six, and seven year old child thins down and becomes more active.[16, 26] This is the time when he is most susceptible to any growth disturbance. These are the ages when Legg-Calvé-Perthes syndrome and nutritional disturbances manifest themselves. Some show growth arrest lines about the metaphysial portions of long bones. Endocrine disturbances, infantile paralysis, acute exanthemas and many other childhood diseases affect growth in a general way. These effects may or may not persist. They are always threatening.

Preadolescents

At approximately 10 to 11 years of age a difference is noted in boys and girls, the girl stepping ahead of the boy in weight and height.[30, 35]

This is the age when precocious sexual development must be watched for. Delayed sexual development will pose for the orthopedic surgeon multiple problems, such as slipped upper femoral epiphysis, congenital coxa vara with severe symptoms, and rounded back or kyphosis with scoliosis.

Adolescents

Examples, as illustrated, show the great range of development encountered at this age. Body build and extent of secondary sex changes are the clues to the internal environmental quickening of steroid growth factors. Chronologic age is an indeterminate factor.[18] Bone age, as determined by hand and wrist x-ray plates, must be the basis in this group for disclosing the individual's true or maturing age.

A Special Child

The cerebral palsied child needs a particular word because of the great demand for adoptable children. This year it has been estimated

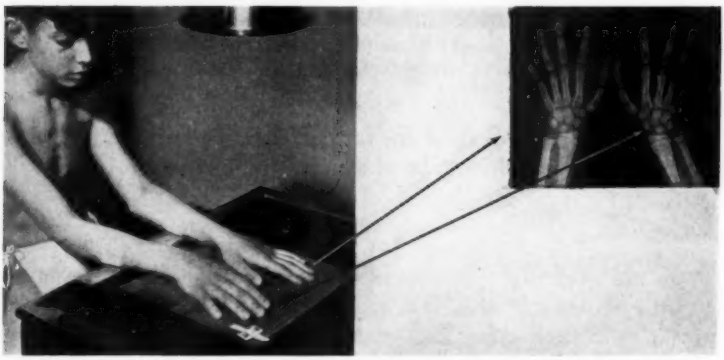


Fig. 4. Position when maturation x-ray plates are taken for determining bone age. (Reproduced by permission of Charles C Thomas, Springfield, 1954.)

that 100,000 babies and small children will find foster parents. The pediatrician is required to "certify" these children prior to adoption. Too often the palsied child is not recognized at an early age.

The cerebral palsied child should be actually diagnosed by six months of age. It is not always easy. Infants vary so in the development of their central nervous system at birth that they frequently retain many acutely sensitive reflex reactions well into their second year. Birth data are often emotionally loaded and of small value as to difficulty of delivery.

A child's motor pattern can be observed, particularly coordination of upper extremities compared to lower extremities, or left as compared to

right. A spastic infant will show a great overflow of muscular reactions when the examiner's hands are sharply clapped over its head. The face may show grimacing, and the tongue may roll from side to side, with a tendency to hyperextend the head and neck. A mild opisthotonos may be present, and involuntary movements are general, the child rarely crying during examination.

The child that can sit up or stand and walk offers but little difficulty. At that stage all the signs previously mentioned are exaggerated, including gait and speech. The Moro reflex persists.

IMPORTANT COMPONENTS OF GROWTH

Proportions of Growth[21]

Parts of the body grow more rapidly than others; the head increases two to two and a half times over its birth mass, and has reached most of this growth by six years of age. The torso increases three times, the upper extremity four, and the lower five times by 18 years of age. Growth continues far beyond most illustrated tables. Army anthropologists clearly proved that the growing plateau is not reached until age 21 in males, and is maintained for two or three years, followed by an appreciable deterioration from then on.[25] This time span is the longest required by any of the primates. Man's growth gradient is unique.

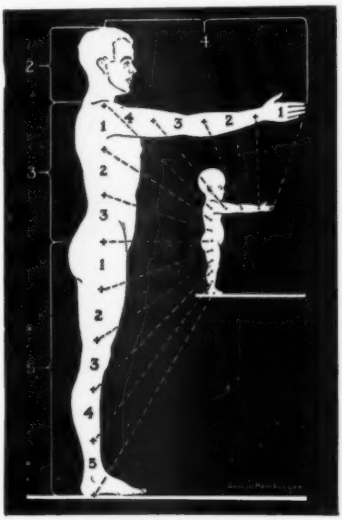


Fig. 5. From birth to maturity the head increases twice its original size; the trunk 3, the arms 4 and the legs 5 times. (Reproduced by permission of Charles C Thomas, Springfield, 1954.)

Motor Development

The Babinski and the sucking reflexes are present before birth. Developing motor responses seem to proceed from the general, as expressed by mass movements of the child, to the specialized and more complex, in progressive development. Coordinated movements proceed from head to trunk to lower extremities. To produce the Moro reflex, the examiner startles the infant by clapping his hands or banging on the examining table. Normally the reflex response consists in drawing up the legs and extending

the arms to embrace with grasping hands. It is considered basically a defensive response, enabling the small one to cling to the mother for protection.

Muscles

Arey believes that shortly after the end of the second month of intrauterine life the fetus may have developed sufficient muscular-nerve structure so that spontaneous movements are possible. At birth 23.4 per cent of the total body weight consists of muscles; 32.6 per cent at 15 years; and 44.2 per cent at 16 years. Puberty increases muscularity, suggesting one reason for a predominant interest in physical activity. Strength, an important aspect of physical development measured by dynamometers, usually as a grip test, increases steadily as the child grows older. Boys are usually more advanced at all ages over girls, especially rising at and after 14 years.

GROWTH AND DEVELOPMENT

Normal Time Table[23]

At *one month* the infant lifts his head, makes swimming movements when he is placed face down, turns his head from side to side, and stares.

At *two months* he smiles.

At *four months* he holds his head raised a little.

At *five months* he rolls over.

At *six months* he sits in a corner of his crib, and grasps well.

At *10 months* he creeps.

At *12 months* he stands and walks with help, holds a pencil in his hand as in writing, and loses the Moro reflex. The mean age of walking for boys is 13.5 months; for girls, 13.6 months.

At *15 months* the child walks alone and has good coordination.

At *18 months* he climbs, throws, scribbles.

At *30 months* he goes up and down stairs alone, tries to stand on one foot, runs easily.

Height and Weight[3, 27]

Growth changes of these dimensions render only general impressions of individual development, whereas body proportions are quite significant. These change at a relative rate for the unaffected child, ultimately achieving adult proportions in steady stages. Certain organs are unique. For example, the nose is small and flat in the newborn,

growing to full adult size earlier than other facial parts. Such natural but apparently disproportionate maturing of a part may cause adolescents great distress, who see themselves resembling Cyrano de Bergerac or Jimmy Durante.

Variability is greatest in weight and the transverse diameters.[33] Individual stature curves based on anatomic or bone ages are more varied and complex than curves based on chronologic ages. Nevertheless, bone age is the best indicator of maturity.[18] Rapidly growing children show the greatest individual variations in many measurements, and estimates are less reliable during these periods. Time bombs of growth go off at different intervals, producing this uncertainty.[27]

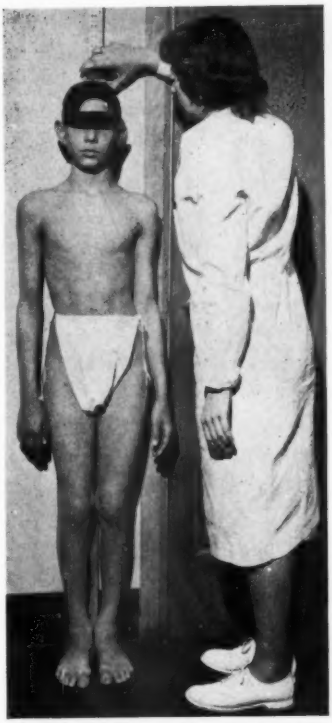


Fig. 6. Technique of measuring stature. Heels, buttocks, back, shoulders and occiput should touch flat wall surface. (Reproduced by permission of Edwards Bros., Ann Arbor, 1952.)

Trunk

The long trunk of childhood occasionally distresses parents. An explanation reassures them that they have not spawned a dwarf. Boys are heavier as a rule and may learn to walk later than girls because of the disproportions of trunk and legs. Short legs are clumsy, while slender legs are better coordinated and provide better leverage. Rapid growth during the preschool years usually reduces markedly this disproportion. Just prior to puberty these changes are greatest, the chest expanding to become more square than triangular. The hip width, bi-iliac diameter expands rapidly, and the waistline shifts. With adolescence all body lines are changed. The boy masculinizes (muscularizes), while the girl develops softer curves and finer, less rugged proportions.

Legs and Feet

These take size commensurate with the child's somatotype or hereditary body build. They vary from long, slender, graceful, to short,

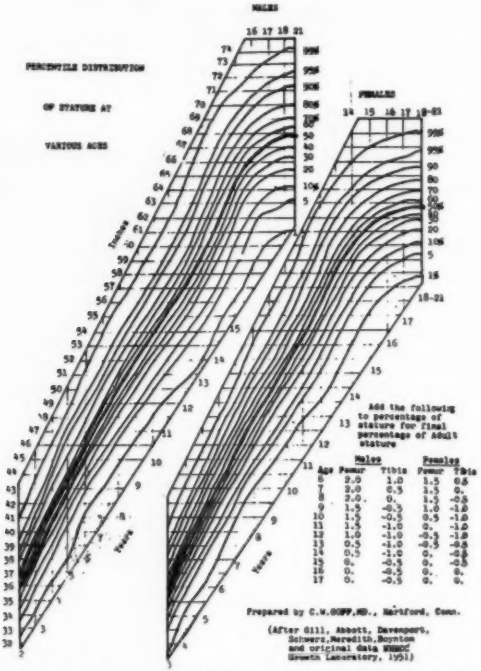


Fig. 7. Percentile charts, males and females, of stature distribution at various ages. The corrections for age and bone of lower extremities are used in the Gill and Abbott formula for appraising expected growth. This is a useful chart because the percentile channels may be used somewhat after the manner of a growth and development channel or graph. (Reproduced by permission of Clinical Orthopaedics and J. B. Lippincott Company.)

stubby and clumsy. Most rapid growth occurs during the first two years, to the horror of the parent, who must buy new shoes every three to four months because the child literally "outgrows" them. At puberty feet also outgrow the rest of the body and seem outlandishly big. Present day desire for similarity in dress among school ages is disconcerting but not hopeless. While the average youth is distressed not to wear "loafers," a careful explanation will usually evoke enough self-interest to get him to wear well lasted, leather soled, laced oxfords that are supportive and protective. Children must be taught to take pride in their shoes and to polish them. Shoe trees for little shoes are a good investment.

Hands

Nail nibblers are common and need cause no anxiety. This distressing behavior usually disappears spontaneously because of self-criticism

when comparisons are made with other children. Pride is a powerful adjunct when properly invoked.

Middle Childhood

This is the time when physical growth jogs along pretty much at an even pace. Rapid gains in weight and stature have taken place in earlier years and will do so again during adolescence. Girls, maturing about two years earlier than boys, often are puzzled over the difference and feel out of step because they seem older than boys of their age.

Rhythm of Female Growth[2, 22, 31]

Most dimensions, including stature and sitting height, show four phases of alternating rapid and slow growth. There is a rapid increase during infancy and a slow, regular rate during childhood. A second accelerated phase begins slightly prior to age 10 and continues for four years, to gradually taper off at about 17 years of age. Thus girls are shorter than boys from three months to 10 years and from 14 to

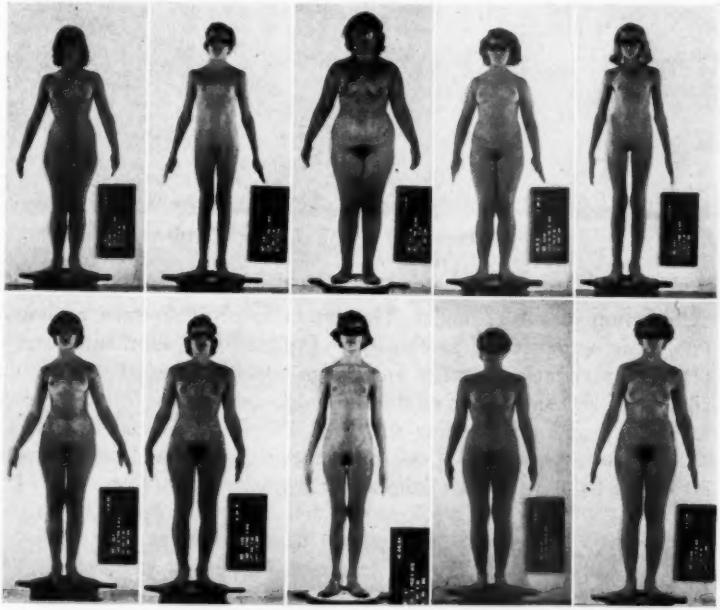


Fig. 8. Range and rhythm of maturity, female. Note the age and development of secondary sex traits, always on a highly individual basis, probably genetically controlled and environmentally influenced.

maturity. Girls surpass boys in stature and sitting height from 11 to 14 years, the crucial steroid growth phase. They also show greater thigh girth from three to 18 years. Girls increase in skin and subcutaneous fat during the same period, exceeding boys readily. Girls show earlier and greater fluctuations of growth during adolescence. This correlates well with the 6:1 ratio of incidence of idiopathic (growth) scoliosis. Terminal stature is attained by girls between 14 and 17 years of age. Menarchal age averages 12.5 chronological years and 13.0 skeletal years.

Rhythm of Male Growth[31]

The first rapid cycle occurs between birth and two years of age. In the next phase, two to 11 years, slowing is apparent. The tempo picks up at 11.5 years for most boys and continues to ages 15 to 18. A plateau or leveling off comes at age 18, lasting to full maturity at 19 to 21 years.

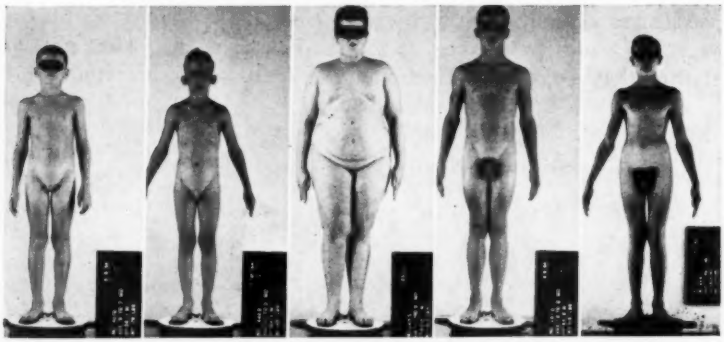


Fig. 9. Range and rhythm of maturity, male.

Deceleration in percentage growth over-all is accompanied by less deceleration in sitting height. This seems to correlate with adolescent idiopathic scoliosis. Measurements of growth at short intervals, if practical, make refinement of knowledge possible, especially concerning adolescent acceleration as related to endocrine balance (steroid growth variations) and approaching maturity. Disorders associated with the 11 to 18 year period, of which there are many, are more apt to come into early focus with such follow-up ratings.

Physiologic Maturity[31]

In girls, breast development begins, on the average, as a protrusion of the nipple and its surrounding areolar area at age 10.6 years, reach-

ıng a round fullness of adult maturity at 13.9 years. Pigmented pubic hair first appears on the average at the age of 11.6 years, reaching a full adult appearance, with the top of the escutcheon horizontal, at an age later than 13.2. The average age at menarche is 12.8 years. Boys show their secondary sex traits at approximately age 11.8 years and

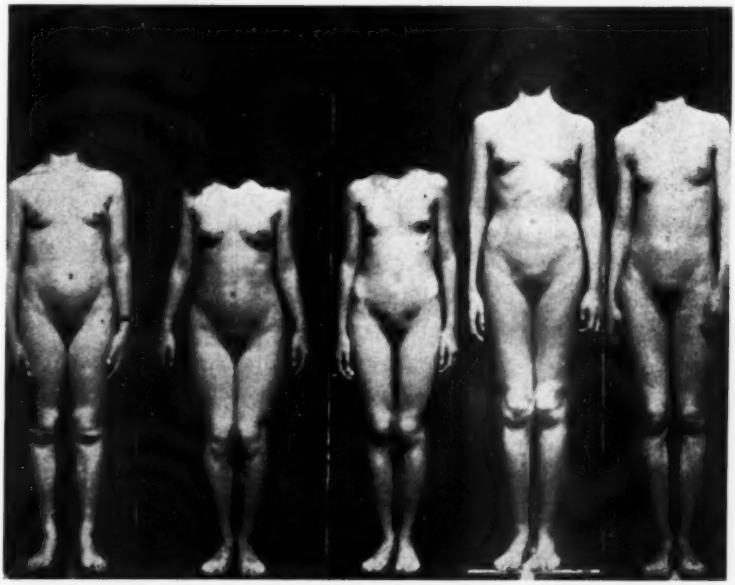


Fig. 10. Range of age of onset of menarche. All children began their menarche on this date; the far left is age 10.5 years, ranging to the far right, whose age is 15.0 years. (Reproduced by permission of Society for Research in Child Development, 1951.)

reach full sexual maturity at 15.2 years. The average age at spermatogenesis is difficult to rate, but is thought to be 14.0 years.

Growing Tensions

Long bones grow, producing tension on fascia, periosteum, muscles and other less dense structures. These may cause discomfort to many children, and are probably among the causes of "growing pains," so frequently encountered. Other causes are, no doubt, strains of a traumatic nature on tension epiphyses, such as the tibial tuberosity in Osgood-Schlatter's disorder and the tuberosity of the os calcis, known s Sever's disease. Greater tensions on pressure epiphyses are thought

to be responsible for some vascular changes within these epiphyses, as in Legg-Calvé-Perthes syndrome.[11]

Fig. 11. Right femoral stimulation following a spiral fracture. Photograph 24 months after closed reduction, with differential between extremities of 2.6 inches.

Asymmetry of tensions may cause irregular growth, as in scoliosis and at the distal epiphysis in amputations. Such irregular growth is observed in defects of the neural arch in all growing ages. Contractures, partial paralysis as in poliomyelitis, may cause deformations of growth. A child with poliomyelitis has a general growth retardation which may disappear in six to 18 months, but the over-all delay is such that frequently the child fails to catch up with his or her former percentile. If a poliomyelitis patient is allowed up early in braces, a good growth response is the rule. But if prolonged nonweight bearing is permitted, growth is delayed.

Local Growth Stimulation[1, 5]

Remodeling of bone after an acute bone infection or a fracture produces a stimulation of growth. Foreign body objects, screws, ivory bone pegs and blocking the medullary cavity with a calcium plug are reported to show growth stimulation of the particular bone involved in children with sufficient growing time remaining. This increment is not great. In the author's experience 1 cm. of growth increase is the maximal achievement, and this has been acquired during the immediate six months following the operative procedure. A splinter, transverse osteotomy will produce a greater increment.

Local Growth Retardation[15]

Height and weight are the best indicators of growth status and the growing process, and records may be quickly obtained. Skeletal age assessment from a hand x-ray plate is necessary to determine with accuracy a child's maturation.[38] These may be recorded on the child's history by a medical technician. An advance or a retardation of growth is immediately apparent when these three dimensions are spotted in the data sheet (Fig. 12). A child should remain relatively within his or her percentile channel if all is well. Deviations are immediately apparent as they are plotted. While this is not as comprehensive as Wetzel's grid, nevertheless, for a routine system it can prove satisfactory and simple of accomplishment.

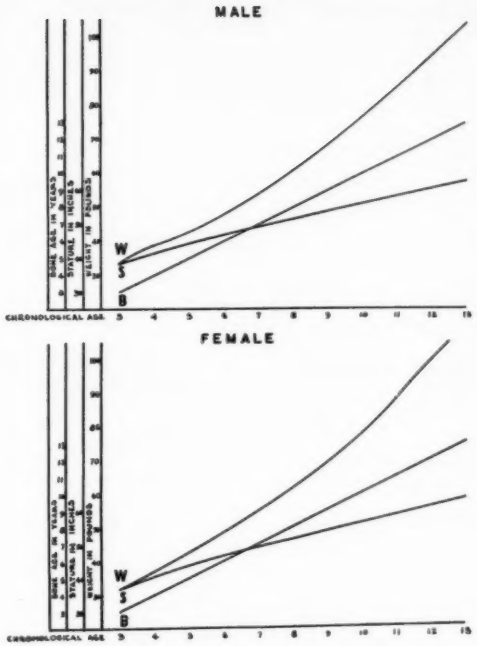


Fig. 12. The anthropological graph, used at Newington Home and Hospital for Crippled Children to plot a child's growth status. The heavy lines are the means for *W*, weight, *S*, stature, and *B*, bone age. (After Simmons, 1944.)

Basal Metabolic Rates

Important information may be obtained by a series of carefully done basal metabolic rates. The young child is handled with difficulty, and may be tested as to his tracer uptake,[37] using I^{131}. Ten microcuries given orally show a normal uptake of 30 to 40 per cent in 24 hours.[28] In this regard there is a sex difference.[7] During the age span six to 17 years males increase in basal oxygen consumption 60 per cent as against females, 30 per cent. The examiner's attention is directed to a probable stimulating effect of certain steroid hormones in the male. Males are, accordingly, operating at a higher metabolic level. Protracted illness, long orthopedic procedures and severe trauma may precipitate a thyroid deficiency.

The excretion of creatine is variable in all age groups and not dependable as a determination of growth increment.

Plasma alkaline phosphatase activity is closely related to growth rate and also to osteoblastic activity in bone metabolism, the ebb and flow

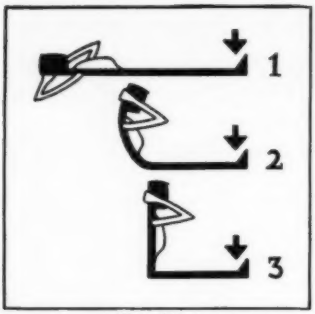
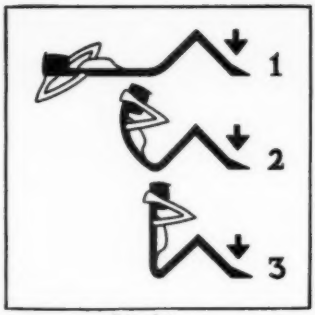


Fig. 13.



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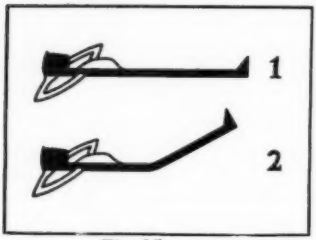

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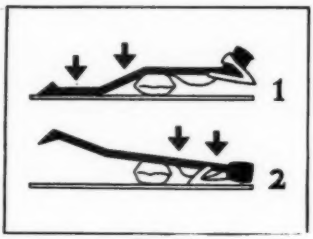


Fig. 16.

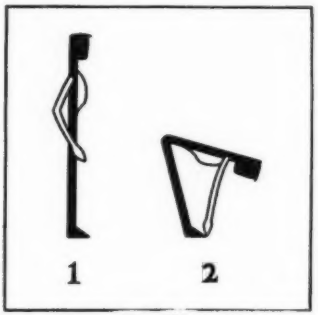


Fig. 17.

Fig. 13. Test exercise 1. Purpose: To test for and to strengthen the upper and lower abdominals as well as the psoas muscles.

Position: Lying on the back, hands clasped behind the neck, feet held down on the table or floor or placed under a heavy piece of furniture as resistance. Keep the hands behind the neck and try to roll up into a sitting position.

Fig. 14. Test exercise 2. Purpose: To test for and to strengthen the abdominals alone.

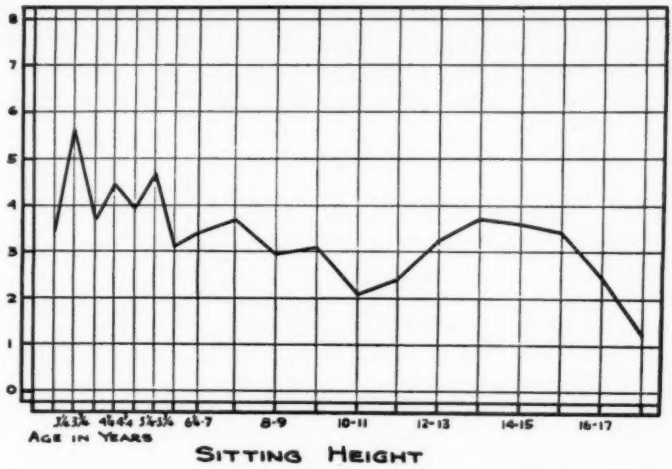
Position: As in Figure 13, but with knees bent. Keep the hands behind the neck and try to roll up into a sitting position as before.

Fig. 15. Test exercise 3. Purpose: To test for and to strengthen the psoas and lower abdominals.

Position: As in Figure 13, with hands behind the neck, the legs extended. Keep the knees straight and lift the feet 10 inches off the table. Keep them there for a count of 10 seconds.

Fig. 16. Test exercise 4. Purpose: To test for and to strengthen the upper back muscles.

Fig. 18.



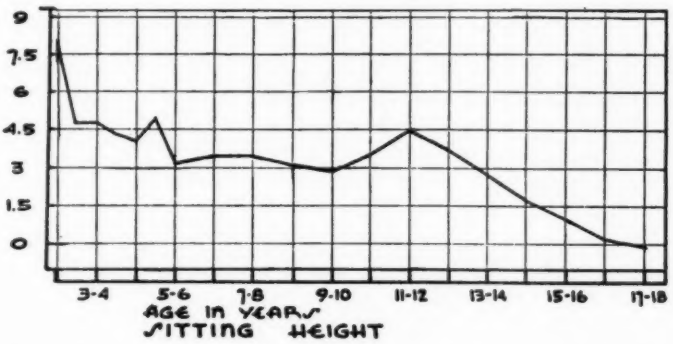


Fig. 19.

Fig. 18. Curve of percentage increase, white males. These mean percentage rates of increase in sitting heights show continued growth well beyond age 17. This correlates with increased rate of idiopathic (growth) scoliosis during the adolescent ages.

Fig. 19. Curve of percentage increase, white females. Although the rate of idiopathic (growth) scoliosis is greater among females, the percentage increase in sitting height is less. This is accounted for by the combined growth deceleration effects of the female steroid hormone and the stimulating growth effects of other hormones, also operating during adolescence, apparently at a more intense rate in the female.

Position: Lying on the face with a pillow under the abdomen (be sure the pillow is large enough), but far enough down to give the body the feeling of being a seesaw so that, if one end were weighted, the other would be able to be held in the air. Then rock back and forth slowly.

Fig. 17. Test exercise 5. Purpose: To stretch the back and hamstring muscles and to test for flexibility

Position: Standing erect in stocking or bare feet, hands at sides. Put the feet together, keep the knees straight, and lean down slowly. Stay down as far as possible for a count of 3. Do not strain, but relax at the end of the stretch.

(Figures 13 to 17, inclusive, reproduced by permission of the Connecticut State Medical Journal, 1955.)

of the bone tide. In infancy these values are high—8 units—and drop off by the second year of life to 6 to 7 units. This plateau is maintained to the ninth year in girls and the eleventh year in boys, when there is a rapid drop toward 2 to 3 units, reached at age 17 in both sexes. Boys, as a rule, have slightly higher values than girls throughout life.

These higher values of serum alkaline phosphatase show good correlations with stature, sitting height and length of upper and lower extremities. In other words, correlations are high where growth is greatest, expressed as in linear measurements. Breadths are not correlated.

ORTHOPEDIC SIGNS OF DISORDERS OF CHILDREN

Gout

That children, especially the adolescent, may be gouty and occasionally show a full-blown syndrome is well known. Multiple joint signs may suggest further investigation with the blood uric acid determination the diagnostic proof.

Pituitary Dwarfism[14]

Facial features are wrinkled and infantile, and body proportions are nearly normal but diminutive. Axillary and pubic hair is absent. The voice is childish; intelligence appears retarded, but usually is nearly normal. Bone structures appear normal, but gait and joint movements are not smooth in action.

Hypothyroidism

An athyroidal child will be retarded or decelerated in all growth dimensions, but especially in bone age, determined by maturation studies of the hands and wrists. The cretin never forms normal growth centers. They are spotty on the roentgenogram. Other classical signs aid in distinguishing this deficiency.

Adrenogenital Syndrome

The height may be retarded. Hyperplasia, on the other hand, may not alter the growth rate. If marked, there are likely to appear signs of virilism, an increased maturation rate and growth in general with an early epiphysial closure leading to a diminished stature, well below the median age.

Froehlich's Syndrome and Obesity

The association of obesity with delayed sexual development or with cryptorchism often leads to a mistake in diagnosis of Froehlich's syn-

drome. Obvious constitutional types of the fat or endomorphic body builds are lumped together and usually called Froehlich-like. This fallacious idea is so prevalent among physicians that it seems important to crush it at this point by briefly outlining what Froehlich described.

Froehlich's syndrome is marked by a diabetes insipidus, real sexual infantilism and eunuchoid proportions of growth. As originally described, such children appear relatively normal during childhood. As they approach 10 to 12 years of age, with the adolescent spurt of growth in the offing, they begin to manifest marked eunuchoid proportions. They gain weight rapidly. Diabetes insipidus is often the real clue to the disorder. X-ray studies will show a definite calcified suprasellar cyst. Only in true cases should the diagnosis or descriptive name "Froehlich's syndrome" be applied.

Taste Blindness and Diabetes[29]

Children who are diabetic are usually retarded in all growth determinations. Of these, 15.3 per cent have been reported as taste blind to phenyletheocarbamide. There is reported some generic association between these two conditions, probably representing a recessive mendelian trait. Children of diabetic parents may be taste tested. If they are blind, their growth factors should be more carefully observed for deceleration.[34]

Myopia and Growth[6]

Boys six to 14 years of age who are myopic are usually 10 pounds heavier and 1.5 inches taller at their mean age. Girls are much less apt to show this growth stimulation until they reach the menarche. At that time they too will go ahead of their mean age group. The mechanism operating is not apparent.

A-Bomb of Hiroshima and Nagasaki

The Stanford University group, headed by W. W. Greulich, report retardation of height, weight and skeletal age, even as late as five years after the bombing. The factors operating are complex, but only one of them, that of the bombing itself, is reported.

Syphilis

X-ray diagnosis of metaphysitis, dactylitis and a positive serology are necessary for a diagnosis.

Rickets

Signs do not appear until the child is three or four months old. Craniotabes is usually first noted, and x-ray studies of long bones show classical cupping of the lower radial epiphyses and others. The costosternal rosary is a rarity these days. Transverse lines of density must not be confused with rickets. These are usually lines of so-called growth arrest and are found much more frequently than formerly believed.

Scurvy

As an orthopedic disorder this rather slowly developing condition is seen less often these days. Tender shins, emaciation and classical fusiform x-ray shadows of long bones are present.

Injury to Peripheral Nerves

Erb's Type of Paralysis. Flaccidity of the upper arm, abduction and internal rotation are present. The Moro reflex is absent, usually unilateral in the newborn.

Klumpke Type. There is flaccidity of the forearm and hand, contracted pupil and upper lid droop on the same side, usually unilateral.

Whole Arm. There is complete flaccidity, easily detected, the reflexes are lost, the muscles are wasted, and there is always considerable anesthesia of skin areas.

Injury to Central Nervous System

Cerebral Edema. The Moro reflex is absent, but returns after a few days.

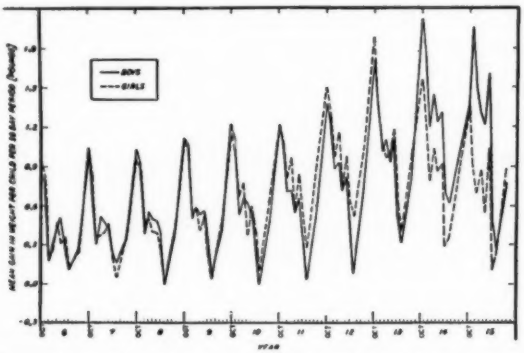


Fig. 20. Seasonal variations of average monthly weight gains, greatest in October, least in May (male and female). (Figures 18, 19, 20, reproduced by permission of the Society for Research in Child Development, 1951.)

Cerebral Hemorrhage. The original Moro reflex may be present, but disappears in a few hours or days after birth.

Cerebral Palsy. This is responsible for as large a number of handicapped individuals as any other single disorder except poliomyelitis. Even poliomyelitis is a form of spinal palsy. Cerebral disturbances leading to a palsy are divided between a flaccid, a spastic and an athetoid state of the nerve-muscle control systems. Associated disabilities, such as impairment of sight, hearing, speech or mental capacity, together with a convulsive state or epilepsy, must be noted and measured with due consideration for any combination or association with other diseases of the bone-muscle-nerve locomotor mechanisms.

SUMMARY

Emphasis must be placed on the growth dynamics of the child. These have been outlined and need no further mention. The orthopedic examination overlaps the pediatric examination at every step. This team play is readily acknowledged. Nobody works in a vacuum.

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DORSAL WEDGING ROUND BACK IN PREADOLESCENTS

ALBERT B. FERGUSON, JR., M.D.

This entity is also known as kyphosis juvenilis, Schuerman's disease or vertebral epiphysitis. Although mild degrees of the deformity are symptomless, it can progress to the point of definite adolescent and adult disability, both functional and cosmetic.

In reviewing cases of this type some information has come to light which may be of help in preventing this deformity. The object of this paper is to promote notice of the anterior vascular grooves in the dorsal spine in six to 11 year old children who have posture problems.

ETIOLOGY

The dorsal kyphos that we all have to some degree has been related to the mechanical stress of an upright posture, the assumption being that an increased load would cause greater curvature (i.e., excessive body weight).

When a child bends forward to touch fingers to toes as in the usual school exercises, it is apparent that fixation of the pelvis by tight hamstrings will cause excessive flexion in the dorsal spine (Lambrinudi).

The so-called Schmorl's node or herniation of the intervertebral disk into the body has been implicated by Schmorl as a cause of progression in the presence of increased stress, the cushioning effect of the disk being lost with resulting increased pressure on the vertebral body. It is true that some of the round back deformity takes place in the disk as well as in the wedging of the vertebral body.

Irregular ossification of the epiphyses of the vertebrae themselves has

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been implicated as a primary cause of the deformity. This type of ossification has been ascribed to circulatory interference or to infection.

Cases of early wedging round back may be seen before the appearance of the epiphyses as an ossifying growth center.

It is intended to call attention to the part played in round back deformity by persistent anterior vascular grooves. This view was originally elaborated by Ferguson in 1939.

NORMAL SPINE DEVELOPMENT

In reviewing normal unselected lateral chest films in the age group from six to 11 years certain observations were apparent.

There is a normal dorsal kyphos, the curvature of which takes place in the area of the fifth, sixth and seventh dorsal vertebrae. The time of development of this kyphos varies considerably, some youngsters still having a straight dorsal spine at age 10.

The anterior vascular groove which is originally present in lumbar as well as dorsal vertebrae is closed in over 60 per cent of the children reaching six years of age. The wedging that develops in the fifth, sixth and seventh dorsal vertebrae to form a so-called normal kyphos results in closure of the grooves in the upper dorsal area.

By the time the age of 10 is reached only 17 per cent have some vestige of a groove remaining. Seventy-one per cent have developed a round back in the fifth, sixth and seventh dorsal vertebrae area. The vestigial grooves are below this area in the dorsal spine. The lumbar spine grooves have long since been closed.

The indentation in the anterior border of the vertebra seen by the roentgenogram has been found by dissection to be occupied by a large endothelium-lined vascular lake formed by the confluence of veins at this point. There is some cartilage also present in the younger children.

If the soft tissues are removed from the vertebral body, the groove depth also will vary, depending on the degree of closure.

RELATIONS OF GROOVES TO PREADOLESCENT KYPHOSIS

Wedging tends to close the grooves in the area of normal dorsal kyphos (D5-D6-D7), and the lumbar grooves usually close early in life. The area where persistence of this entity may take place is in the mid-dorsal spine.

Further wedging of these vertebrae added to the normal dorsal kyphos may be sufficient to produce an accentuated round back deformity. The deformities appear to develop by the adding on of vertebrae below the normal kyphos area.

A group of 21 round back cases that had follow-up examinations of

Fig. 21. Fig. 22.

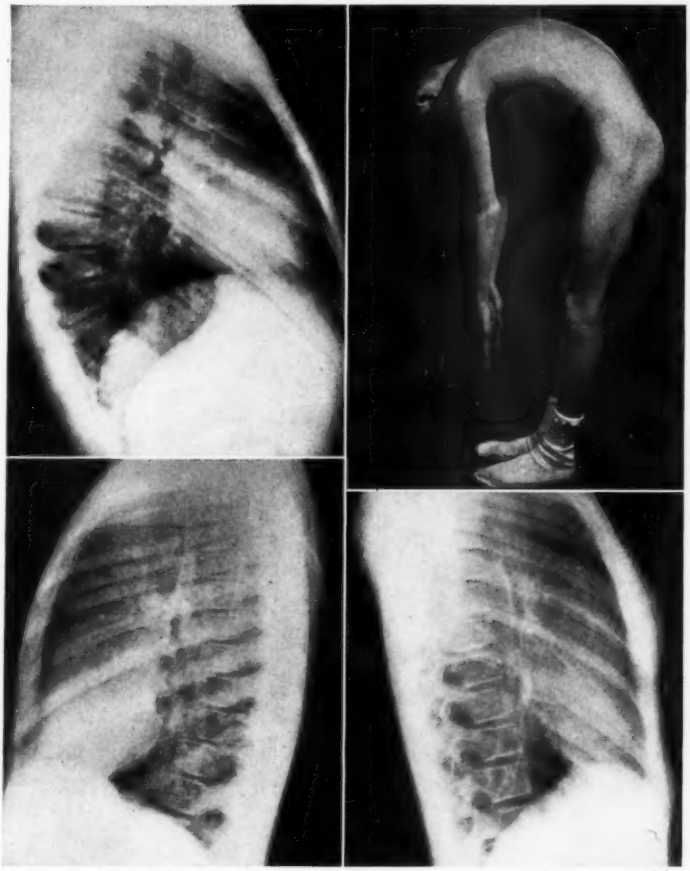

Fig. 23. Fig. 24.

Fig. 21. Established dorsal wedging round back deformity in a 15 year old boy. The vestige of a persistent vascular groove at the apex of the dorsal curve is pointed out by the arrow. Secondary changes such as irregular ossification of the vertebral plates are seen.

Fig. 22. The limitation on forward bend caused by tight hamstrings is seen. The dorsal spine tends to flex excessively in an attempt to gain further bend.

Fig. 23. A 7 year old boy showing normal dorsal spine development. The anterior vascular grooves are closed except for a vestige at D5, D6 and D7, where slight wedging has taken place.

Fig. 24. Normal spine development in a 10 year old girl. Grooves are closed, and there is slight wedging of D6, resulting in a normal degree of dorsal kyphos.

Fig. 25. Fig. 26.

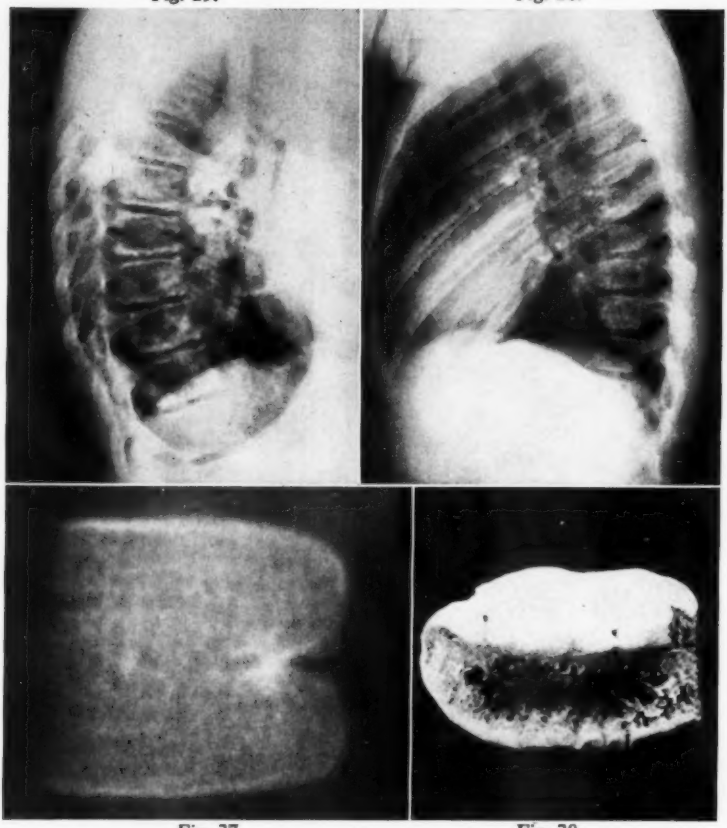

Fig. 27. Fig. 28.

Fig. 25. Accentuated round back as wedging below the D5 to D7 occurs. Some of the grooves are still persistent. (Boy, age 13.)

Fig. 26. Accentuated dorsal round back in the preadolescent is shown (girl, age 8 years 10 months). The anterior vascular grooves are still persistent.

Fig. 27. Roentgenogram of vertebral skeleton. The anterior vascular groove has closed in one area with condensation.

Fig. 28. Vertebral skeleton showing area occupied by persistent anterior vascular grooves.

one to nine years were examined by roentgenogram. Eighteen of these showed progression.

The area involved by vascular grooves was noted and a line drawn parallel to the superior border of the most proximal and inferior border of the most distal.

The angle formed by these lines was then divided by the number of

vertebrae involved to get an average figure for each vertebra. It should be pointed out that the amount of wedging possible in the disk has not been separated out from that in the body itself. The exact same area was measured later at the time of the follow-up film and the average increase calculated. This tended to be about 2 degrees per vertebra.

Not all vertebrae progressed, only a high percentage. Some grooves closed without the slightest wedging, and yet the tendency appeared to be there always, if the load was great enough.

Eight cases of well established round back deformity were seen. Seven of these showed evidence that vascular grooves had been persistent. This consisted either of a slight remnant, hardly enough to mechanically affect the back, to a dense line in the central section of the vertebra interpreted as being due to overriding of trabeculae.

Over 14, the epiphyses, which had begun appearing in the nine to 10 year age group on the female side, tended to become irregular in their ossification if the vertebra was markedly wedged.

Should the anterior border become osseous even though the groove persisted centrally, it would be apparent that a point of mechanical weakness had been strongly reinforced.

This pointing out of the tendency to wedge of vertebrae with persistent grooves does not enter into the controversy regarding epiphysitis of the spine or other etiologic factors noted particularly in teen-agers. The age when the persistent grooves appear to play their most significant role is in the six to 10 year old group.

These grooves do not in themselves constitute the only cause of a dorsal wedging round back. It is felt, however, that they constitute an area likely to collapse under the influence of stress. The upright posture is stress of itself, but habitual slumping and sitting attitudes may increase this strain.

TREATMENT

The child who appears to be developing an excessive lumbar lordosis and an accentuated dorsal round back posture and who is then found on a lateral film of the dorsal spine to have wide-open anterior vascular grooves becomes the object of special consideration. Preventive measures which may be of aid in relieving dorsal spine stress include the following: sleeping in a stretched out position—not "curled up like a ball"; elimination of a pillow; use of a bed board; 20 minute periods of relaxation flat on the back on the floor; postural exercises designed to obliterate the lumbar lordosis; elimination of forward bending exercises; stretching of tight hamstrings; well defined heels on the shoes.

Measures beyond these include a spinal "flexion" brace, wedging

body jackets and even rarely fusion in an area of deformity and pain which is progressive. These measures would be used of course on those patients afflicted with a severe condition. The degree that this deformity can reach would amaze those not regularly dealing with it.

SUMMARY

A review of unselected lateral chest films and those with a dorsal round back tendency has been conducted. This reveals that the area of normal dorsal kyphos is dorsal five to seven. Progression of wedging has been found in those vertebrae with persistent anterior vascular grooves which adds mid-dorsal vertebrae to the area of normal dorsal kyphos.

Evidence that anterior vascular grooves have been persistent has been found in cases of well established adolescent kyphosis. Irregular ossification of the epiphyses is felt to be secondary to the altered stress of a wedged vertebra.

Children in the six to 10 year age group who have dorsal round back posture problems deserve roentgenograms of the dorsal spine and especially firm consideration should open anterior vascular grooves be demonstrated.

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PRONATION IN CHILDHOOD

FRED R. ZEISS, M.D.

The problem of the pronated foot is being given high priority in the thinking of the pediatrician and general practitioner. At the 1954 meeting of the American Academy of Pediatricians, 82 per cent of the members attending the round table discussion of the orthopedic problems of children requested that the problem of the pronated foot be considered This paper is an attempt to answer the many questions put to the panel, Dr. W. T Green and the author, at that time. No attempt will be made to discuss the rigid or spastic flatfoot, nor will the valgus foot of paralytic origin be included, nor any of the congenital fixed deformities such as equinovarus, metatarsus varus or talipes calcaneus. Pronation is the common foot abnormality which becomes apparent as soon as the infant begins to stand, and calls for recognition and treatment.

ANATOMY AND PHYSIOLOGY OF THE FOOT

A review of the anatomy and physiology of the foot will help to lay the foundation for the discussion to follow. At birth the baby's foot is made up of almost equal parts of soft tissue and bone. This proportion changes rapidly as the child grows, so that by the time growth is completed these values are changed to 90 per cent bone and 10 per cent soft tissue. In the newborn the os calcis, astragalus, the metatarsals and most of the phalanges are ossified. The navicular, cuboid and the cuneiforms are usually cartilaginous, and are soft and pliable. With the exception of the cuboid these are the bones that make up the highest segment of the long arch. In the mechanics of weight bearing they are subjected to great stresses and strains, and are molded by these forces.

The long arch is situated on the medial side of the foot and is composed of the os calcis, astragalus, navicular, cuneiforms and first,

second and third metatarsals. These bones articulate in such a fashion as to strike an arc. This archlike arrangement absorbs shock, lends strength, elasticity and rhythm to the stride, and makes possible a smooth transfer of weight from the hindfoot to the forefoot, and from one foot to the other. If the bones of the long arch are distorted in size and shape, the transmission of power through the foot is impaired, and efficiency is lost.

The transverse arch of the foot is not as apparent as the long arch and is best demonstrated by placing both feet together. It is then seen that there is a gradual slope across the foot from the base of the fifth metatarsal upward and medially to the base of the first, where the apex of the transverse arch is reached.

All the bones that make up the arches are intricately interlaced with ligaments of tremendous strength. The plantar surface of the foot is further reinforced by the tough spring ligament and the long plantar ligament. These ligaments play an important role in maintaining the stability of the foot. The foot is supported by both static and dynamic forces. The bones and ligaments provide static support, and the muscles dynamic support. The anterior and posterior tibial and the peroneous longus muscles function importantly in maintaining the arches of the foot. The tibial muscles are inserted into the bones which make up the highest segment of the long arch, and their tonic power reinforces the arch from above. The peroneus longus muscle swings beneath the lateral malleolus into the plantar surface of the foot across to its insertion into the base of the first metatarsal, slinging the long arch from below and preventing the transverse arch from splaying out. The intrinsic muscles of the plantar aspect of the foot supported by the plantar fascia function to truss up the arches from below.

The foot is extremely mobile. Most of its motion takes place in either the extreme proximal or distal portion. Also the medial aspect of the foot is more mobile than the lateral. Dorsiflexion and plantar flexion of the foot take place at the ankle joint. Inversion and eversion and supination and pronation occur mainly at the subastragalar joint, with some slight motion added by the midtarsal joints. Adduction and abduction occur in the midtarsal joints. Flexion and extension of the toes take place at the metatarsophalangeal and the interphalangeal joints respectively.

The foot supports the body weight in standing and in walking. In standing, balance is essential; in walking both balance and propulsion are required. In standing the foot supports the body weight largely through the bones, ligaments, and tonic power of the tibial, peroneal and gastrocnemius muscles. In walking the body weight is propelled by the action of practically all the muscles of the lower extremity. Walking is a complex function and calls for the same high degree

of muscle coordination as does speaking or swallowing. In analyzing the mechanism of walking it is seen that a combination of hip, knee, ankle and foot motion takes place in sequence. In stepping off, the hip and knee are flexed, the foot dorsiflexed; then the knee is extended, and the posterior aspect of the heel strikes the ground. Now the hip is extended, and plantar flexion of the foot begins. The weight is transferred into the foot distally along its lateral plantar aspect through the os calcis, cuboid, base and shaft of the fifth metatarsal to its head, and then medially across the metatarsal heads to the first metatarsal-phalangeal joint (Fig. 29). At this point, by a combined powerful contraction of the peroneus longus and gastrocnemius muscles, the take-off thrust is delivered. When the take-off is completed, the shift of the weight to the opposite foot begins. Any distortion in size and shape of the bones, relaxation of the ligaments, loss of power of the muscles or disturbance of nervous control upsets this smooth flow of power and makes for inefficiency, awkwardness and symptoms.

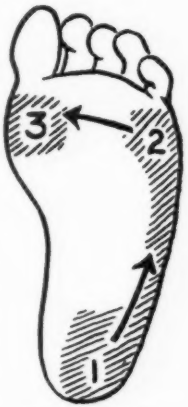


Fig. 29. Plantar surface of foot showing weight-bearing areas and sequence of transmission of weight through the foot. (Courtesy of Dr. Stuart A. Thomson.)

The foot is probably the hardest-worked part of our anatomy and next to the hand the most dexterous. It tolerates ordinary abuse well. It inherits many defects, is frequently injured, and occasionally becomes the target of disease. Most of us make our living standing upon our feet. It is our responsibility to protect the foot and to nurture its proper development so that when full stature is attained the individual has adequate feet to stand upon. This will not only increase his physical well-being, but also enhance his economic welfare.

ETIOLOGY

In most children pronation is an inherited defect, prenatal in origin, a family or racial characteristic. It is frequently seen as a part of an over-all picture of an undue amount of congenital ligamentous relaxation in which hyperextension of the elbow, wrist, fingers, hips, knees and ankles is present. These children are frequently called "double jointed" (Fig. 30). Opposed to this condition, pronation is also seen in children who are born with posterior structure tightness, especially tight heel cords. It is only by abducting and everting the foot that they are able to get their heels to the ground. This throws the stress of weight bearing on the medial side of the foot and causes pronation.

In a smaller percentage of children pronation is acquired. It is caused

by debilitating illness, prolonged bed rest, immobilization in casts. There is another group of children whose feet become pronated because of faulty sleeping, sitting and walking habits. Dr. J. H. Kite has repeatedly reported these faulty activities. The popular face down or stomach-sleeping position almost universally advocated by pediatricians usually forces the lower extremities into external rotation, the foot into 90 degrees of out-toeing, and rests the weight of most of the lower extremity on the medial aspect of the great toe and first metatarsal, a

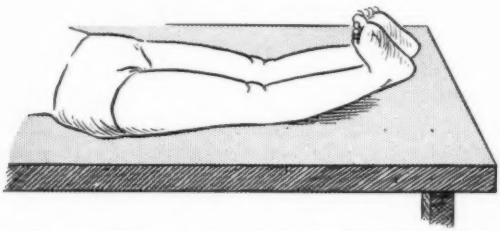


Fig. 30. Congenital ligamentous relaxation permitting hyperextension of knee and excessive dorsiflexion of feet.

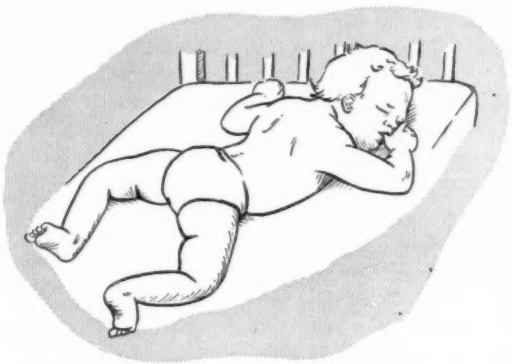


Fig. 31. Faulty sleeping position.

part of the foot poorly designed to take stress (Fig. 31). The medial ligaments become stretched and the soft midtarsal bones improperly molded. The baby who continues to walk with a wide stance does the same thing (Fig. 32). The teeter-babe, the kiddy-car and the walker may also be offenders. The child, to propel these vehicles, is forced to push off from the medial side of his foot. Television has developed a generation of floor sitters. Many of these children sit back on their legs, forcing their knees to become knocked and their feet pronated (Fig. 33). In children six to 10 years of age the development of an

accessory tarsal navicular bone may cause the foot to pronate or may account for lack of improvement in a foot being treated. The enlargement of the accessory navicular changes the point of insertion of the posterior tibial muscle and its direction of pull. Its distorted pull rotates the navicular downward and produces eversion of the os calcis.

To summarize, then, prenatal influences, heredity, congenital ligamentous relaxation, congenital posterior structure tightness, poor sleeping, sitting and walking habits, debilitating illness, obesity, and the development of accessory tarsal navicular bones are factors in the production of pronated feet.

Fig. 32.

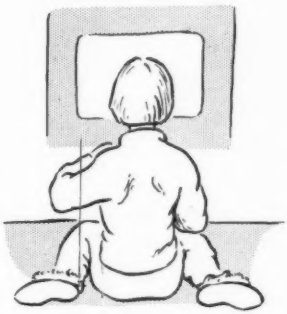


Fig. 33.

Fig. 32. Faulty stance—persistence of wide base.
Fig. 33. Faulty sitting position.

OBJECTIVE FINDINGS AND SYMPTOMS

Objective findings are listed first because pronation is recognized long before the baby is capable of complaining. It may be diagnosed as soon as the baby begins to stand and may be suspected even earlier. This foot is flexible and exhibits no fixed deformity; contours are normal and may even possess a well developed arch when not weight bearing. However, when the baby stands, a metamorphosis occurs; there is downward rotation of the astragalus and navicular (Fig. 34), eversion of the os calcis (Fig. 35), abduction of the forefoot and obliteration of the long arch (Fig. 36). The medial border of the foot bulges and may take on the shape of a rocker, and the forefoot angulates on the hindfoot at the most prominent part of the bulge. This is the "Dr. Jekyll and Mr. Hyde foot" so aptly described by Thomson.[21] As the

medial border of the foot gives, secondary changes may develop higher up in the lower extremity. The medial malleolus falls closer to the floor, the knees swing into valgus (knock knee), and the thighs become adducted (Figs. 37, 38). The weight-bearing alignment of the lower extremity is altered so that an imaginary plumb line dropped through the center of the patella will fall medial to the inner border of the foot and not through the second toe as is normal.

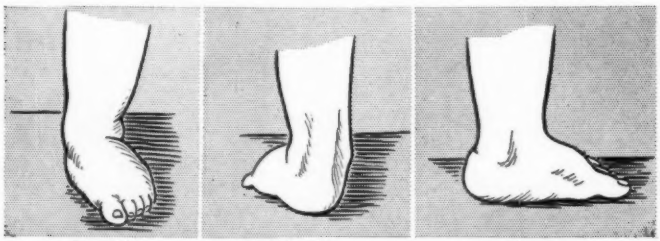


Fig. 34. Fig. 35. Fig. 36.

Fig. 34. Downward rotation of the astragalus and navicular and abduction of the forefoot.
Fig. 35. Eversion of the os calcis and swinging out of the heel cord.
Fig. 36. Obliteration of the long arch.

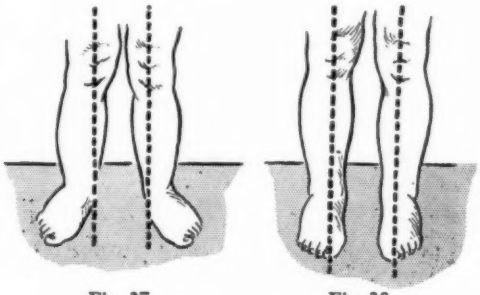


Fig. 37. Fig. 38.

Fig. 37. Distorted weight-bearing alignment.
Fig. 38. Normal weight-bearing alignment.

Many of these children walk late, are awkward and unstable and fall unduly. They frequently exhibit gait defects. Some of these children toe out, while others toe in. Out-toeing is the more undesirable gait because it forces the child to take off from the medial side, the weak side, of his foot and aggravates the abnormality. More of these children with pronation toe in, are pigeon-toed or ding-toed. This is the gait that disturbs the parents. The toed-in gait is the child's attempt to protect the weak side of his foot by voluntarily throwing his weight to the lateral side. It is a compensatory gait and should not be dis-

couraged. Reassurance should be offered the parents. As the foot increases in strength, the toeing-in diminishes. The pernicious practice of providing an outer sole wedge or a special toed-out shoe to break this habit should be discouraged.

Subjective complaints include fatigue, desire to be carried, sore feet, pains in the calves and about the knees, and muscular cramps which usually come on shortly after the child has fallen asleep. Subjective complaints are not common and are seen much more frequently in the child who toes out. Because some of these older children find it difficult to compete successfully in group activities and athletics, they may become introvertive. They tend to resort to the radio or television for their relaxation and recreation. This lack of exercise may further aggravate their foot weakness and obesity.

EXAMINATION

Completeness demands that the patient be examined in three positions, lying, standing and walking. In the recumbent position an

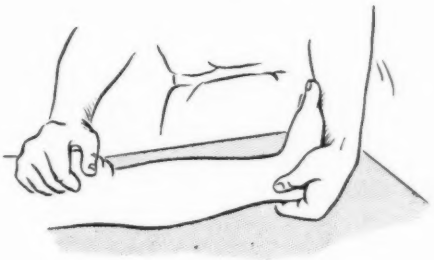


Fig. 39. Tight heel cord.

estimate of the general build and physique of the patient is made. The tone of the muscles and ligaments is appraised, leg lengths and contours noted. Knee and ankle jerks are elicited. The range of motion of the hips, knees and ankles is tested. Especial attention is given to the tightness of the heel cord.

Many cases of tight heel cord will be overlooked unless the test is carried out in the following manner. With the patient supine and the knee completely extended, the foot is grasped by the heel so that the sole of the foot falls into the examiner's hand and plays up volar aspect of his wrist. It is then slightly inverted and dorsiflexed (Fig. 39). In this maneuver the foot should easily come up to 30 degrees above neutral. Anything less than 30 degrees indicates tightness of the heel cord. If care is not taken to invert the foot so as to lock the heel, the heel swings into eversion as the foot is dorsiflexed, and little if any

tension is placed on the heel cord. A muscle evaluation is helpful and can be completed rapidly in the cooperative child by asking the patient to hold against the examiner's pull in dorsiflexion, plantar flexion, inversion and eversion.

The standing child is inspected both from the front and from the back. He stands in his bare feet, which are held about 6 inches apart and toed straight ahead so that their medial borders are parallel. From the front the medial bulge of the inner border of the foot, the downward rotation of the astragalus and navicular, the abduction of the forefoot and the obliteration of the long arch are easily seen. A knock knee is immediately noted and its degree estimated. A simple and accurate measurement for the record is to determine the number of fingers that separate the medial malleoli with the medial femoral condyles barely approximated. The greater the distance between the malleoli, the more severe the knock knee.

The position of the heel can best be evaluated from behind. By following the direction and course of the heel cord to its insertion into the os calcis, the position of the os calcis is revealed. In the normal foot the heel cord drops perpendicularly into the os calcis, but in the pronated foot, because of the eversion of the os calcis, the heel cord swings out or laterally, describing a letter C. The more severe the eversion, the greater the outward curve. The downward drift of the medial malleolus and the plantar rotation of the astragalus and navicular are also better seen from behind.

Observation of the walking child, wearing his shoes, completes the examination. The child with a normal gait toes straight ahead, walks with a heel-toe stride, has a good take-off, coordinates the swing of his arms with the motion of his legs, and appears graceful. The child with the pronated feet may toe in or out, walks with the entire foot striking the floor at the same time, a flat stride, lacks take-off and may throw his legs laterally in an attempt to clear his knock knee. This makes him appear awkward and poorly coordinated. Examination of the patient walking barefooted is valueless. The floor may be cold, the feet tender, and the patient unaccustomed to this practice, bringing many protective mechanisms into play which are wholly unnatural.

Lastly, examination of the shoes may assist in analyzing a foot problem or a gait defect. Abnormal wear on the heels and soles, broken down counters and misshapen shanks or vamps are points to be observed and evaluated.

DIAGNOSIS

The diagnosis of pronation is made on the objective findings of a flexible foot which exhibits no fixed deformity when at rest, but, when

weight bearing, exhibits an obliteration of the long arch, downward rotation of the astragalus and navicular, eversion of the os calcis and slight abduction of the forefoot.

In the differential diagnosis the hypertrophied plantar fat pad must be considered. In the baby who is just beginning to stand and take his first steps, the hypertrophied or large plantar fat pad may simulate a pronation because the fat pad obliterates this baby's long arch. Here the likeness ends. There is no eversion of the heel or abduction of the forefoot, and the astragalus and navicular occupy a normal position in the long arch. Also there will be no distortion of the weight-bearing alignment of the lower extremity. Equinovarus, metatarsus varus and the talipes calcaneus are fixed deformities. The valgus foot of paralytic origin is often thick and pudgy and is associated with paresis of muscles, absence of reflexes and lack of sphincter tone and control. The spastic foot usually exhibits an equinovarus deformity and is accompanied by increased muscle tone and increased and pathologic reflexes. The cavus foot is differentiated by its high arch, the clawed toes and tight plantar fascia.

TREATMENT

Support is the keynote of treatment! Adequate, continuous, courageous and steadfast support is essential from the moment the diagnosis is made until the pronation is cured or the foot is full grown. A delay of several years in initiating treatment to determine whether or not the patient will improve spontaneously is inexcusable. Valuable time is lost, time during which the pliable midtarsal bones may become deformed and the medial tarsal ligaments overstretched by the stresses of weight bearing. Statements occasionally made to parents, indicating that most children are flatfooted, or that once a flatfoot always a flatfoot, are erroneous and are not substantiated by long-term follow-up. Persistent, uninterrupted and adequate treatment improves all pronated feet and cures many. Exercises, control of obesity, and correction of faulty sleeping, sitting and walking habits complete the regimen of management.

Shoes

Until the infant stands, any covering for the foot that will keep it warm is adequate. However, when the baby begins to stand, support is necessary. A high shoe with a flexible shank and a firm sole should be provided. Some brands of baby's shoes contain a rigid shank which almost functions like a splint, preventing the baby from working and developing the muscles of his feet. These are to be avoided. Even the

plump, heavy baby will be adequately shod in a flexible shanked shoe. It is well to keep the child in a high shoe until his heel is sufficiently developed to hold an oxford type of shoe. The roughened, calloused heels and heel cords often seen in youngsters are due to the sawing of these soft tissues by the hard counters of a slipping low shoe. Also some of the gait defects seen in youngsters wearing low shoes are due to an attempt on the part of the child to hold his shoes on with his toes.

The pronated foot is treated as soon as the baby stands by prescribing a standard high shoe with a ⅛-inch inner heel wedge. If the child toes out unduly, it is well to extend the wedge into the sole. The wedge overcomes the eversion of the heel and gives some support to the long arch by throwing the weight to the outside of the foot. The wedge should be checked by the physician to see that the alterations have been done properly. Inexperienced cobblers frequently tack the wedges on the wearing surface of the heel, and in a few days the wedge loosens, may cause the child to fall, or is torn off completely. The heel wedge should be cut so that it is the same shape as the heel. It is fashioned so tha tthe medial border is ⅛ inch high and tapers to a knife edge on the lateral border. The heel of the shoe is then opened up, the wedge inserted, and the heel resewn (Fig. 40). If a sole wedge is used, it should be fashioned and inserted in a similar manner.

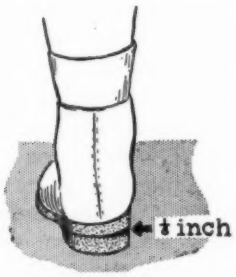


Fig. 40. Wedge properly inserted, carrying all the way across to the lateral border of the heel.

When the child is walking well, his long arch can be more adequately supported by the wearing of an insert or arch support. This insert is made so that it closely fits the foot and supplies maximum support at the point most needed. The ⅛-inch inner heel wedge neutralizes the eversion of the heel, the insert supports the astragalus and navicular and prevents their downward rotation, and the high shoe applies the foot snugly to the corrections. This makes for an ideal support program. Inserts are custom-made by most orthopedic supply firms. They are made up of a leather exterior stuffed with felt, rubber or cork, depending upon the physician's desire and the peculiarities of the firm making up the support. A steel arch support may be desirable in a very heavy person or an extremely weak foot. These are more difficult to fit and more difficult to wear.

Most orthopedic firms require a prescription designating the height of the elevation and a tracing of the bare, weight-bearing foot showing the location of the first metatarsal-phalangeal joint and the medial malleolus, and a marker showing how far laterally the elevation is to

be carried. The elevation prescribed varies from ¼ to ½ inch, depending upon the size of the foot and the weight of the child. In the early walker ¼-inch elevation is sufficient; in the large child over eight years of age ½ inch is not too much (Fig. 41).

Children accept an insert immediately. Seldom is a breaking-in period required, and soon they become so accustomed to their supports that they seldom go without them. Inserts need not be renewed with each change of shoe and may be transferred from one pair of shoes to the next until worn out or outgrown. All our children wear them at least six months, most nine months, and occasionally a child can wear them a year. Water deteriorates this type of insert rapidly, and if the child gets them wet, they should be removed from the shoe, dried in the air, rubbed until the leather becomes pliable, and replaced.

The orthopedic shoe that comes equipped with the Thomas heel is seldom prescribed. It is too heavy and too rigid, and the built-in long arch support is usually inadequate, and not properly placed. The child has no opportunity to exercise his feet, and it is like asking a child to wear walking casts. A child who toes out unduly can be assisted by the provision of a pronator shoe, a special shoe built on an intoed last. For the children who toe in, the pronator shoe is not prescribed because it may aggravate the intoeing.

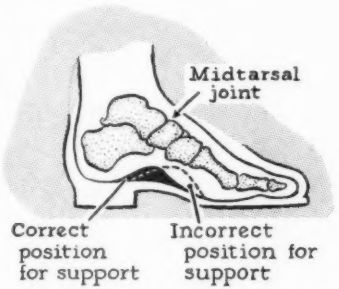


Fig. 41. Correct position for support beneath the midtarsal joint. Insert should reach its maximum height at level of navicular. (Courtesy of Dr. Stuart A. Thomson.)

This support program need not necessarily be an expensive treatment. Once the regimen is established, the child is checked at intervals of six months.

Exercises

Exercises play an important part in the treatment of the pronated foot. The cooperation of both the patient and the parents must be secured to carry out an efficient exercise regimen. Practically, it is extremely difficult to get both. However, a combination of support and exercise is the ideal treatment. If the exercises are not done, the patient is only partially treated.

Graduating from the simple to the more complicated, the exercises consist in supination, toe curling and gastroc setting exercises.

The simplest supination exercises consist in picking up objects with the toes. A number of marbles are placed within easy reach of the

right foot, and a pail is placed off to the left, barely within reach of the right foot as the child sits on a low chair. The patient is taught to pick up these marbles one at a time with his toes and transfer them from the floor to the bucket. When this is accomplished, the process is reversed, the marbles are placed within easy reach of the left foot,

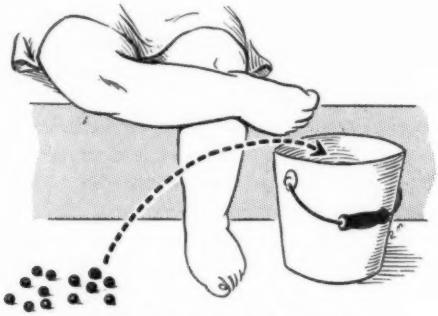
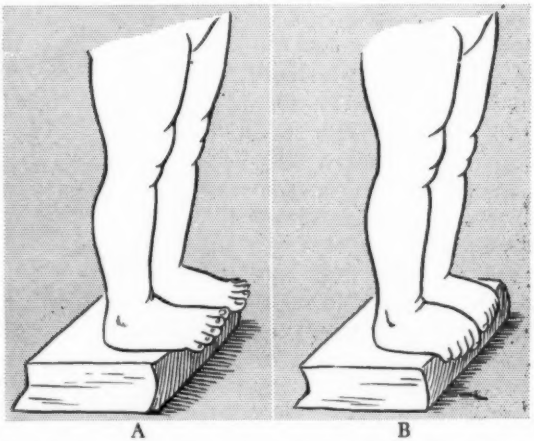


Fig. 42. Simple supination exercise.



A B

Fig. 43. Toe curling exercise. *A*, First position. *B*, Second position.

and the bucket is placed on the right. A 10 minute picking-up session twice daily is very helpful (Fig. 42).

Older children may be taught to walk on the outer border of their bare feet with their toes curled in. This should be done on well carpeted floors; otherwise the outer borders of the feet become so tender that little is accomplished. Toe-curling exercises in which the patient grasps an overlapping edge such as a stair, chair rung or large book to

the count of three and then relaxes for three counts are helpful (Fig. 43).

Lastly, gastroc setting exercises may be useful. The patient is taught to stand on one foot and raise his body weight on his tiptoes 25 times, four times daily; this is to be carried out alternately with both feet.

If the heel cord is tight, active and passive heel cord-stretching exercises are prescribed (Fig. 44). If the peroneal muscles are tight or overpull, the inversion and abduction exercises described by Dr. J. H. Kite are helpful.

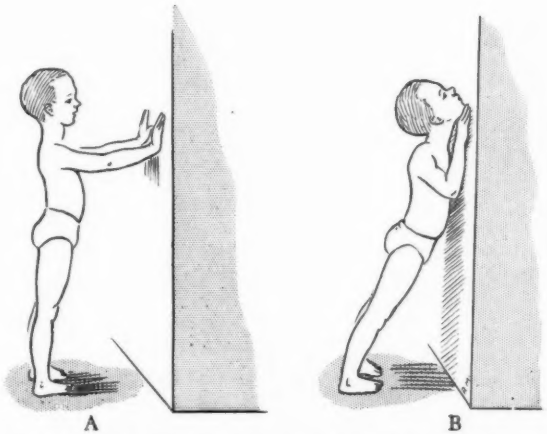


Fig. 44. Active heel cord-stretching exercise. *A*, First position. *B*, Second position.

Other Measures

Correction of faulty sleeping, sitting and walking habits is also indicated. In the full grown, symptomatic pronated foot which is disabling, surgery may be considered. Several adequate and satisfactory corrective bony operations have been described.

CONCLUSION

The pronated foot is not something the child has to live with. It should not be a cross he is forced to bear from childhood into adult life. Pronation can be successfully treated! It is our responsibility to recognize it early and treat it adequately. In so doing more people will be brought to adult life with strong feet, better enabling them to meet the demands of our highly competitive existence. Less and less will we hear the all too common complaint, "Doctor, my feet are killing me."

SUMMARY

The importance of early recognition and early treatment of the pronated foot is emphasized. A review of the anatomy and physiology of the foot has been furnished. The symptoms, objective findings, routine of examination and diagnosis are discussed. A practical, inexpensive program of management has been outlined.

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METATARSUS VARUS

Recognition and Treatment

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In any discussion of metatarsus varus the question arises whether this entity should be separated from metatarsus adductus. Two articles in American orthopedic literature are entitled "Congenital Metatarsus Varus." Peabody[6] called the more severe and more treatment-resistant feet showing this type of deformity, metatarsus varus. Altman and Hanisch[1] agreed with this opinion of Peabody's. They stated that the metatarsus varus deformity was difficult to correct and that the metatarsus adductus deformity was easily corrected. Kite[3] called the milder and more easily corrected examples of this deformity metatarsus varus, while he described the more difficultly corrected form metatarsus adductus.

This complete reversal in terminology is confusing. Furthermore, this terminology is based on difference of degree of deformity and a difference of degree of correctability. There is no clear-cut anatomic basis for such a division of terminology. Until there is made available an anatomic basis for the division of terms, only one term should be used. I have chosen to use the term "metatarsus varus."

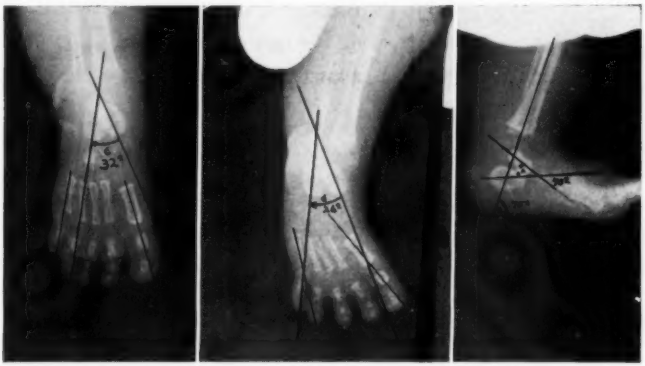
It is customary to describe a deformity of an extremity by noting the abnormal relation of the more distal part to the more proximal part, or that part which is closer to the center of the body.

At present there is no unequivocal evidence presented that abnormal tibial torsion, either internal or external, is associated significantly with the deformity of metatarsus varus.[2, 4] The relation of the astragalus to the tibia is considered to be normal.

DESCRIPTION OF THE DEFORMITY

Hindfoot

In metatarsus varus, depending on the degree of its severity, the proximal group of foot bones, or hindfoot, consisting of the talus and the calcaneus, may or may not be abnormally oriented one to the other. The normal arrangement on physical examination is for the vertical axis of the calcaneus or heel to parallel the vertical axis of the leg between the upper and lower ends of the tibia. In other words, the calcaneus should be aligned in the weight-bearing axis of the leg. It should also be able to be moved from side to side.



a b c

Fig. 45. Metatarsus varus, left foot. *a*, Foot at 6 months, clinically corrected. There is a suggestion that the midfoot abduction was not corrected; since the forefoot, although correctly aligned with hindfoot, is displaced laterally, the long axis of the talus running far medial to the first metatarsal rather than through it or through the first interspace. *b*, *c*, Foot at 3 months, when treatment was begun. These roentgenograms were made with the foot in a weight-bearing position.

When the normal relation exists between the talus and the calcaneus, roentgenograms of the foot taken in the anteroposterior view, with the foot and leg in the weight-bearing position, show a 25 to 35 degree divergence of the long axis of the talus and the calcaneus (Figs. 45, 48). In the severe form of metatarsus varus the calcaneus is usually in a rather marked valgus position—in other words, the heel angles outward in relation to the vertical weight-bearing axis of the leg. Anteroposterior roentgenograms of the foot taken in the weight-bearing position when a valgus position of the heel exists show an increased divergence of the long axis of the talus in relation to the long axis of the calcaneus (Fig. 47). In metatarsus varus the angle between the long axis of the talus and the long axis of the calcaneus by roentgenogram may therefore be within normal limits in the mild cases or

increased beyond 35 degrees, usually in the more severe cases (Fig. 47). In the very severe forms, usually associated with other congenital abnormalities, the valgus of the heel may be *fixed* so that the heel is not able to be moved into varus by the examiner.[5]

In the normal foot and metatarsus varus there is no equinus deformity. In other words, the structures posterior to the ankle joint, including the capsule of the ankle joint and the subastragalar joint and

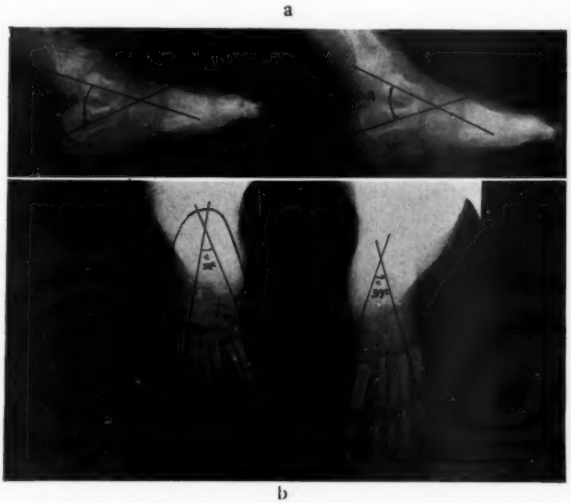


Fig. 46. Metatarsus varus, bilateral, mild, 4 year old boy. *a*, Lateral views. The divergence angle between the calcaneus and astragalus appears normal in the *lateral* view, although these are not weight bearing films, and with weight bearing the angle might increase. *b*, The divergence angles between the long axes of the calcaneus and astragalus in the anteroposterior views are normal. These are standing films. The medial position of the navicular (marked with a cross on the right foot), in relation to the center of the head of the astragulus, is laterally positioned. The first metatarsal axis parallels, but lies lateral to, the long axis of the astragalus. The fifth metatarsal axis points medially and crosses the calcaneal axis.

the Achilles tendon, are elastic. In normal infants and those having metatarsus varus when the heel is held in a neutral position,[3] the dorsum of the foot may be brought upward so that it touches or almost touches the anteromedial aspect of the lower leg.

Lateral roentgenograms taken of the foot and leg in a weight-bearing or standing position show a 30 to 45 degree angle between the long axis of the talus and the long axis of the calcaneus both in the normal foot and in metatarsus varus (Figs. 45, 46, 47, 48). In a clubfoot this angle is markedly reduced (Fig. 49). In this same lateral view in stance the long axis of the tibia subtends angles with both the floor and the

long axis of the calcaneus of less than 90 degrees in the normal foot and in the metatarsus varus deformity (Fig. 48). In the clubfoot deformity in stance the angle between the long axis of the tibia and the long axis of the calcaneus is greater than 90 degrees because of the equinus deformity (Fig. 49).

Midfoot

In metatarsus varus the bones of the middle part of the foot, namely, the navicular, cuboid and the three cuneiform bones, are abducted or angled laterally on the bones of the proximal or hind part of the foot—

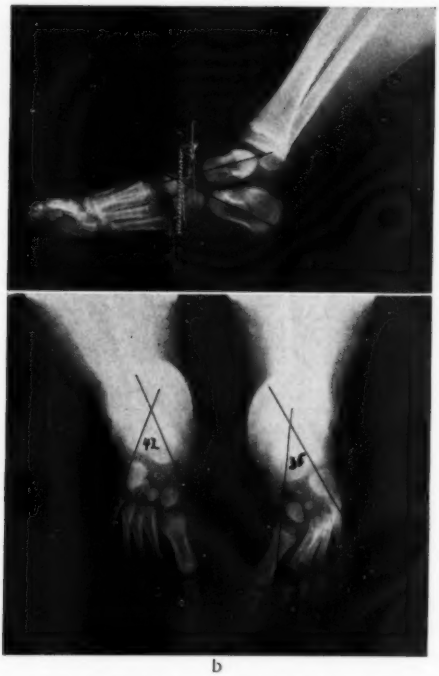


Fig. 47. Metatarsus varus, bilateral, more severe adduction deformity of the left forefoot, 1½ years old. *a*, Lateral view of left foot, although not taken in stance, shows an apparently normal calcaneal-astragalar angle of 40 degrees. *b*, The anteroposterior views are taken in stance. The right foot shows a calcaneal-astragalar angle of 40 degrees. This is above normal and is characteristic of a valgus heel deformity. The left foot showed a much more marked adduction deformity of the metatarsals and a supination deformity characterized by some superimposition of the bases of the metatarsals. The medial angulation of the astragalar head in the hindfoot, the lateral angulation of the midfoot with the lateral position of the navicular (dot mark) and the medial angulation of the forefoot are obvious.

a

b

Fig. 48. Uncorrected metatarsus varus, 8 years old. Despite the deformity which has become fixed during 8 years, the heels show, in both the anteroposterior and lateral standing views, normal calcaneal-astragalar angles. Valgus is not present. There is no equinus, as noted by an 82 degree tibial floor angle in the lateral view. The navicular articulates with the lateral aspect of the astragalar head. The medial angulation of the metatarsals in relation to the laterally angulated midfoot is obvious. The base of the first metatarsal articulates with the medial aspect of the first cuneiform bone, and a hallux valgus deformity is already moderately severe.

the talus and calcaneus. This is obvious in the anteroposterior x-ray view of the foot taken after the ossification centers of the bones of the middle part of the foot have developed (Fig. 47). Characteristically, the cuboid is visible by roentgenogram at birth (Fig. 45). The third cuneiform appears about the first year, the first cuneiform at about three and a half years, and the navicular and second cuneiform about

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the fourth year. Actually, the navicular bone may develop an ossification center as early as 18 months. In any event, if the roentgenogram of a foot with a metatarsus varus deformity is made after these ossification centers have developed, it will be seen that the navicular lies lateral to the midpoint of the head of the talus (Fig. 46). This abduction deformity of the bones of the midfoot is seen not only in a mild case such as is observed in Figure 47, but also in the severe cases[5] (Fig. 48).

The fact that both the midfoot abduction and valgus of the heel (characterized by a divergence angle between the long axis of the calcaneus and the talus near the upper limit of normal—that is, 35 degrees or slightly above) exist in both the mild (Fig. 46) and the severe or corrective resistant varieties (Fig. 47) of metatarsus varus has led us to believe that there is no real anatomic difference, from the osseous standpoint at least, between the severe and mild forms of this deformity. In those cases in which the deformity is allowed to persist even though the anatomic variation is not severe as in the case illustrated in Figure 47, *b* (right) (where the deformity was allowed to exist to the third year), treatment will be difficult. On the other hand, when the anatomic deformity is severe at birth, particularly when associated with a fixed valgus of the heel showing a lack of elasticity of connective tissue structures, treatment will also be difficult. Fortunately, only a small percentage of *newborn children* with the metatarsus varus deformity have fixed valgus of the heel and fixed forefoot adduction, or, in more general terms, fixed connective tissue structures associated with the osseous malposition.

Forefoot

In metatarsus varus the metatarsal bones and digits of the distal part of the foot (forefoot) are adducted or, in other words, angled toward the midline of the body in relation to the tarsal bones of the middle part of the foot (Fig. 50). Furthermore, both the distal and middle parts of the foot are supinated in relation to the proximal part of the foot. As a result, the first metatarsal articulates with the medial side of the first cuneiform bone (Fig. 48), and the apex of the convexity on the lateral side of the foot occurs at the junction of the cuboid and the base of the fifth metatarsal (Fig. 46). This angulation is obvious on an anteroposterior roentgenogram of the foot in a weight-bearing position (Fig. 45, *b*).

If the anteroposterior view of the metatarsus varus foot is taken without placing the foot in a plantigrade position, but simply hung limply over the x-ray plate, the supination deformity of the midfoot and fore-

foot becomes apparent. In such a positioning the metatarsals are seen more or less in a lateral view and will appear curved; if the deformity is great, they and the cuneiform bones of the midfoot will appear more or less superimposed. In a severe and rigid deformity, even in an attempted weight-bearing position, this may be observed. Figure 47, *b* (right), shows this to some extent at the bases of the metatarsals.

This adduction and supination deformity of the forefoot is also observed in the clubfoot or equinovarus deformity. Figure 49 illustrates this angulation in a mild and incompletely corrected clubfoot deformity. However, in the clubfoot deformity the midfoot is *adducted* in relation to the hindfoot, and therefore the navicular lies to the *medial* side of the head of the astragalus rather than to the lateral side as in metatarsus varus. In this way one may differentiate between an adduction of the metatarsals remaining after incomplete correction of the deformities of a clubfoot and the adduction of the forefoot observed in metatarsus varus.[5]

a

b

Fig. 49. Uncorrected equinovarus, 9 years old. The long axes of the calcaneus and astragalus are parallel in the anteroposterior view and form an angle of only 19 degrees in the lateral view. This is characteristic of a varus heel.

a, The midfoot does not angulate laterally on the hindfoot, but rather a little medially, as shown by the fact that more than half of the navicular is medial to the midline of the astragalar head.

b, The adduction deformity of the forefoot is the same as that seen in metatarsus varus with the base of the first metatarsal articulating on the medial aspect of the first cuneiform.

The superior articular surface of the astragalus is flattened.

The physical examination may be correlated with the x-ray findings. When the foot is held limply, the supination of the forefoot will be apparent, as will the adduction deformity of the metatarsals and the toes. Flaring of the toes is frequently observed (Fig. 50). The convexity of the outer border of the foot may be observed with its apex at the base of the fifth metatarsal bone. Where the supination of the midfoot and forefoot is marked, there may be vertical, abnormal creases on the middle of the medial side of the foot, and a higher than normal arch will be seen. (In relation to the high arch and the supination of the forefoot and midfoot, the lateral roentgenograms taken after the epiphysial center of the

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navicular bone is well formed will sometimes show a triangular-shaped navicular bone, the base of the triangle being at the dorsal aspect of the navicular bone toward the top of the foot.)

When the foot is placed in a weight-bearing or plantigrade position and a thrust is exerted downward on the upper end of the tibia toward the foot, an interesting phenomenon occurs in metatarsus varus. In the severe grade of this deformity, of course, even when the foot is placed in a weight-bearing position, the supination of the forefoot cannot be corrected and the heel is already in a valgus position. However, in the

Fig. 50.

Fig. 51.

Fig. 50. Metatarsus varus in the infant, front view. *A*, Plantigrade or weight-bearing position. *B*, Foot allowed to hang limply. Note that supination of the forefoot disappears in weight bearing, but adduction deformity of the forefoot remains.

Fig. 51. Metatarsus varus in the infant, rear view. *A*, Foot allowed to hang limply. Note neutral position of heel. *B*, Foot in weight-bearing position. Note valgus position of heel, which develops simultaneously with correction of forefoot and midfoot supination in Figure 50, *A*.

mild cases, which form the large majority of these deformities, the forefoot on weight bearing will assume a position in which all the metatarsal heads touch the floor. At this point the heel will be seen to be in a valgus position of greater or less degree, depending on the severity of the deformity (Figs. 50, 51). This may occur even though when observed in the relaxed position the heel was neutral in relation to the vertical axis of the leg. It would appear that this simultaneous correction of the forefoot supination and appearance of heel valgus is associated with the "calcaneo-cuboid lock" described by Schwartz and Heath.[7] In other words, there is a supination deformity of the forefoot and midfoot in relation to the calcaneus. When "correction" of the supination of the forefoot is obtained by weight bearing, the "lock" between the supinated portions of the foot and the neutral heel causes the heel to go into valgus. Of course, this can happen only when there is a movable subastragalar joint.

A principle in the treatment of metatarsus varus is also involved in this concept of a "lock" between the calcaneus and the cuboid. The heel, or calcaneus, must be externally fixed in a neutral position in relation to the leg during the correction of the forefoot supination and adduction (Fig. 53). Otherwise a type of flatfoot deformity will develop during the correction. The need for this precaution was pointed out by Kite.[3] Nove-Josserand and Fancillon[5] state that one boy "first treated when 21 months old had a metatarsus varus which corrected satisfactorily in orthopaedic shoes except that after treatment the feet appeared a little flat." In other words, they had successfully corrected the metatarsus adduction deformity, but had not corrected the supination deformity at the calcaneocuboid joint, probably because they could not adequately control the heel in the corrective shoe. Thus, after treatment, the forefoot appeared satisfactory, but the heel was in valgus rather than a normal neutral position in relation to the vertical axis of the leg.

Commonly Used Differential Criteria

Metatarsus varus is distinguishable from clubfoot or talipes equinovarus by two simple clinical observations: First, the foot of a normal infant and the one showing metatarsus varus can be dorsiflexed until the dorsum of the foot almost touches the front of the lower leg. This is not possible in equinovarus because of inelastic posterior ankle structures. Secondly, in the large majority of cases of metatarsus varus the calcaneus can be moved into either varus or valgus, but in those cases of metatarsus varus in which the deformity is severe and fixed at birth or of long standing the heel may be fixed *in valgus* and will resist attempts of the examiner to bring it to a neutral or varus position. In clubfoot or equinovarus the heel will be fixed *in varus* and will be resistant to attempts to place it into a valgus or even a neutral position.

There should be no confusion between the condition of metatarsus varus and a congenital or flexible flatfoot. In the latter conditions, although the heel is in valgus on weight bearing, there will be a *concavity* along the outer border of the foot as opposed to a *convexity* in metatarsus varus (Fig. 52).

Diagnostic Criteria

1. Forefoot and toes are adducted and are resistant to correction in abduction past the midline when the heel is held in a neutral position. This is not variable: it is the essence of the condition.
2. There is a convexity of the lateral border of the foot. This is not variable.

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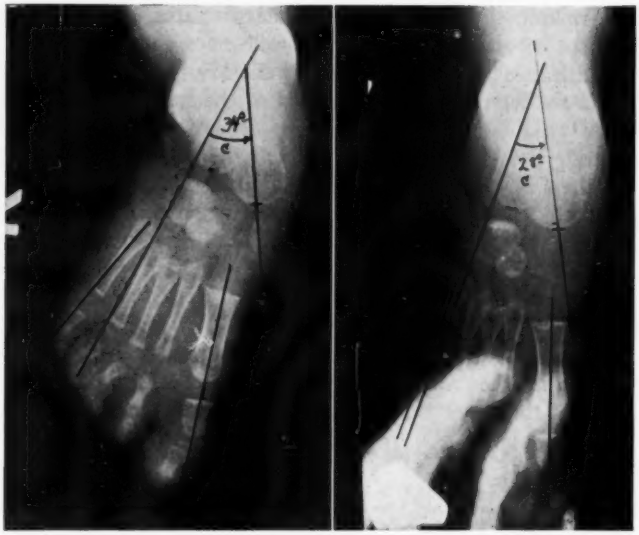

a b

Fig. 52. A flexible flatfoot, 2½ years old. *a*, Right foot in stance; full pronation. Note convexity of medial border of the foot and practically straight line along outer border of foot. The forefoot deviates laterally from the hindfoot axes. The calcaneo-astragalar angle is 34 degrees when the heel is in clinical valgus. The astragalar head appears to slide forward in relation to the calcaneus. *b*, Right foot in stance, but with heel in neutral. The calcaneal astragalar angle decreases to 28 degrees, and the astragalar head appears to move back on the calcaneus. There is still an abduction deformity of the forefoot. The navicular assumes a more central position on the distal articular surface of the astragalus.

3. There is a higher than normal long medial arch. (There may be associated abnormal vertical skin creases on the middle of the medial side of the foot.) This is variable and is related to the degree of forefoot supination.

4. There is a neutral or valgus heel. This is variable.

5. Supination of the forefoot and midfoot in relation to the calcaneus of the hindfoot is also variable.

INCIDENCE

Among 1232 consecutive cases of primary foot diagnoses at the A. I. DuPont Institute, Wilmington, Delaware, Shands[9] reported that 11 per cent were metatarsus varus. In the severe grade there is a hereditary tendency, according to Kite,[3] and there are often associated congenital deformities in other parts of the body, as in the case reported by Nove-Josserand and Fancillon,[5] in which, despite a negative hereditary his-

tory, the child with the most severe deformity which they reported had dislocation of the radial heads at both elbows. In relation to 300 cases of easily corrected congenital metatarsus varus, Kite reported only nine severe cases.

Literature on this subject began to accumulate about 40 years ago, and the early reports are concerned primarily with the severe metatarsus varus deformities. This would give the impression that the mild deformities were only becoming apparent or were increasing in incidence in the last few years. However, this is probably an illusion. For instance, Peabody[6] reported 15 cases. These apparently represented his complete experience. Of the 13 patients he treated, the average age at the inception of treatment was 3.7 years. He found that it was difficult to treat these patients, and he had to use surgery. On the other hand, 17 years later, Kite reported treatment of 300 cases all of which he handled satisfactorily by closed plaster of Paris techniques, and of these, 94 were recognized at birth, and the other 206 cases were seen at an average age of 2.8 months. In view of the rapidity of skeletal growth in the first six months after birth, the difficulty of correction will be greater after this period. Pediatricians aware of this are detecting the deformity early, and as a result the number of reports of resistant metatarsus varus deformities in the literature has progressively decreased over the last 40 years. On the other hand, the majority of the cases seen early are so easily corrected that few orthopedic surgeons see much point in reporting them unless, as did Kite,[5] they report a large number of cases.

Males and females are about equally affected. In over half the cases the condition is bilateral, but, when occuring unilaterally, the metatarsus varus deformity involves the left foot more frequently than the right.

ETIOLOGY

The etiology is unknown. Peabody and Muro[6] suggested that an abnormal attachment of the insertion of the anterior tibial tendon to the plantar fascia or the mesial portion of the transverse metacarpal ligament as observed in one of his own patients (eight years old) might cause the deformity. However, there is not enough evidence obtained by autopsy examinations of deformed newborns that the abnormal anterior tibial insertion is the *cause* of the deformity: it may have developed only during the years since their patient walked with his foot in an abnormal position. Peabody also noted that Bankhart, during operations on the feet of children with metatarsus varus, found some abnormality not clearly described of the anterior tibial tendon attachment. Bankhart suggested that the first cuneiform was missing in meta-

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tarsus varus, but this conclusion is not sustained by evidence collected since he wrote his paper, nor by a specimen from a newborn, the dissection of which I have observed.[8]

DIFFERENTIAL DIAGNOSIS

1. The most common clinical problem is to differentiate a metatarsus varus from a clubfoot, or, in other words, an equinovarus deformity. This differential diagnosis has been carefully made above.

2. *Metatarsus Primus Varus.* In some feet one will see an adduction deformity or medial angulation of the first metatarsal away from the other metatarsal bones. This is apparently due to an isolated abnormal medial angulation of the distal articular surface of the first cuneiform bone. The lateral border of the foot does not show a convexity as in metatarsus varus.

3. *"Toeing-in."* This is sometimes a presenting complaint in metatarsus varus if the child is over one year of age, but may also be due to a lack of normal developmental external torsion of the tibia, or, in other words, persistent internal torsion of the tibia, in which the long axis of *a perfectly normal foot* is pointed medially toward the midline of the body. This is well described by Hutter and Scott.[2]

4. *The Lack of Correction of the Forefoot Adduction Component of the Clubfoot Deformity.* The history should of course rule this out, since there will probably be a story of attempted correction of a clubfoot. The x-ray differences between this condition and metatarsus varus are illustrated by Figure 49, in relation to the other figures.

ASSOCIATED DIAGNOSIS

Some or all components of the metatarsus varus deformity may be seen in which muscle imbalance and/or inelastic soft tissue structures around the joints of the feet are due to (*a*) arthrogryposis (other joints are involved as well as the feet; there is more apt to be heel varus than valgus, or, in other words, a clubfoot deformity); (*b*) Friederich's ataxia (family history and neurologic findings); (*c*) poliomyelitis (when an isolated peroneal muscle weakness exists); (*d*) in some cases, spina bifida (usually a meningocele and characteristic neurologic and x-ray findings).

TREATMENT

The majority of cases of metatarsus varus, a large number of which are now being detected by careful postnatal examination, are rarely associated with congenital deformities in other parts of the body. In

general, this deformity can be corrected in the first year of life before walking begins, and when corrected in this early period the deformity is not apt to recur. In the first week or two of life mild metatarsus varus deformity may be observed. If, upon stabilization of the heel in a neutral position with the leg, the forefoot can easily be swung into an abducted position past the midline, then one may withhold further treatment until the situation is reviewed three to four weeks later (Fig. 53). Some of these cases appear to clear spontaneously.

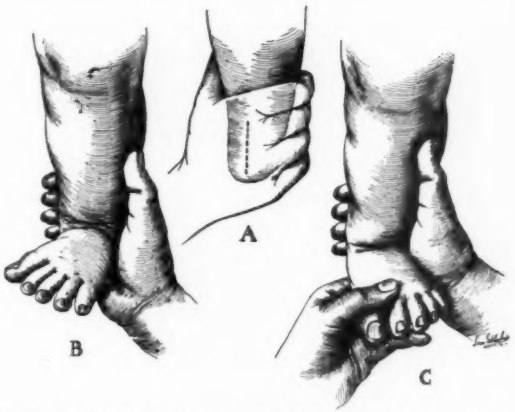

Fig. 53. Correction of the metatarsus varus deformity. *A*, Rear view showing how operator fixed heel in neutral. *B*, Method of stabilizing heel in a neutral position. Forefoot supination and adduction obvious. *C*, Correction of both forefoot adduction and supination by the operator's left hand while the heel (that is, the calcaneus) is maintained in a neutral position.

Some orthopedic surgeons find that in mild deformities a cooperative mother may be taught to hold the heel in a neutral position in one hand and stretch the forefoot steadily for 30 to 60 seconds into the abducted or corrected position several times a day. Such treatment may in the very mild cases be effective. However, if no improvement is noted in three or four weeks, a plaster of Paris cast should be used for treatment. Some orthopedic surgeons use a Denis Browne splint and tape the feet to the splint. This can also be a satisfactory method of correction of a metatarsus varus deformity as long as good control of the heel can be maintained during correction of the forefoot and midfoot deformities.

The plaster of Paris bandage is applied quickly over a minimum of padding, and the foot is then held in a position of correction. The heel is held in a neutral position with one hand, the thenar eminence of which is placed over the lateral side of the heel and the cuboid area while the other hand is placed around the forefoot from its medial side. Pressure is then applied against the medial aspect of the first metatarsal

so that the adduction of the forefoot is corrected in relation to the fixed heel or hindfoot (Fig. 53). Supination of the forefoot and midfoot is usually a factor in the deformity and may be corrected at the same time.

During the first three months of life weekly cast changes are recommended. While in a plaster boot the foot usually becomes more pliable, so that after the second or third plaster change a full correction of the forefoot deformity is possible, even though originally it was fairly resistant with a maintained neutral position of the hindfoot. In a mild case, seen shortly after birth, the correction is usually obtained in about four weeks. In more resistant cases and in those seen later in life the correction takes place more slowly. A slightly overcorrected position of the metatarsal adduction is maintained for three or four weeks after initial correction when the correction has been easily obtained. However, if the patient is seen late or the deformity is resistant, the correction has to be maintained for a much longer period. For instance, if the correction is made at the age of one year, it may be necessary to have the child walk in a plaster boot for 12 weeks or longer after correction has been obtained so that no possible recurrence of the deformity will recur prior to remodeling of the deformed bony structures. Furthermore, when feet must be held for a considerable period in a walking cast so that remodeling can take place in the bones of the feet, they should be held simply in a corrected position.

Kite[5] has pointed out that constant care must be taken to be sure that one does not follow the clubfoot routine during plaster correction: the heel might thus be forced into a valgus position, thus creating a type of flatfoot. Whether or not the plaster of Paris boot extends from the toes to a point above the knee or just below the knee depends on several factors, such as the resistance of the foot to treatment, the amount of adipose tissue on the leg, the contour of the leg and the ability to apply a cast below the knee that will not slip off the foot.

A Denis Browne splint fitted with shoes, the shoes rotated out about 20 degrees on the cross bar, may be used at night to help hold correction of the feet after plaster of Paris treatment until such time as the orthopedist is sure that the deformity will not recur. Once corrected and correction having been maintained for a sufficient period for internal remodeling of the bones of the feet to the new contours, normal shoes may be worn.

A clinical method of checking on the progress of the correction of metatarsus varus is as follows:

The child's foot is placed in a weight-bearing position on a piece of paper, and the outline of the foot is drawn. The convexity on the lateral border of the foot with its apex over the base of the fifth metatarsal can be noted to disappear as successive tracings are made over a period of several weeks. The lateral border of the normal foot describes

a straight line or a slight concavity with the apex of the concavity at the base of the fifth metatarsal over the cuboid bone. At the end of treatment it is advisable to obtain roentgenograms so that a comparison may be made between the pretreatment films and the post-treatment situation. These roentgenograms should be made both in the antero-posterior and lateral weight-bearing positions.

In Figure 48 a metatarsus varus deformity in an eight year old boy is illustrated. At this age the rate of bone remodeling is so slow that repeated manipulative corrections with plaster of Paris retention are no longer feasible. In such situations the forefoot adduction and supination must be corrected by a surgical procedure. The one most commonly used consists of a "dome" osteotomy through the base of each metatarsal. It is then possible, after wound closure, so to position the foot in a plaster of Paris boot that after healing of the osteotomies a satisfactory forefoot alignment in relation to the midfoot and hindfoot components is obtained.

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PARALYTIC FOOT DEFORMITIES

PHILIP D. WILSON, JR., M.D.

A paralytic foot deformity is a fixed contracted attitude of the foot and ankle which impairs the functions of standing and locomotion. The designation is usually reserved for deformities arising in those types of paralysis in which the muscles are flaccid; in other words, diseases involving the lower motor neuron, peripheral nerve or muscle itself.

INCIDENCE AND SIGNIFICANCE

Though a common cause of crippling, paralytic foot deformities are not seen frequently by the average practitioner. In an orthopedic hospital, however, there are many such cases. Some idea of their incidence can be gained from the fact that at the Hospital for Special Surgery over 1300 cases came to operative stabilization in the 12 year period between 1935 and 1947. Probably two to three times this number were treated as outpatients in the same period.[8]

Though the treatment of this condition properly belongs to the orthopedist, it is important that the pediatrician know how to recognize the various deformities early in their course and that he be informed about the diseases in which they are likely to occur. As in most fields of medicine, an ounce of prevention is worth pounds of cure. The paralytic foot must be treated from the beginning, and continual careful observation of the growing child is half the battle won.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

Stance

The bony and ligamentous structure of the foot is adapted to carry the weight of the body in standing and distribute it as evenly as possible

between the *heel* and the *metatarsal heads*. The longitudinal arch, higher at the inside border, is maintained by the shape of the tarsal bones and also by the several strong plantar ligaments. There is really no transverse arch from a functional viewpoint, and each of the metatarsal heads bears weight. This structure, to be efficient, must be stable, and the body weight must be distributed evenly. To prevent unnecessary strain, alignment of the lower extremity joints must fall directly over the midline of the foot. Since the tarsal joints have a certain range of mobility in inversion (supination) and eversion (pronation), stability must depend on proper muscle balance. The muscles providing this lateral balance are the evertor or peroneal group, especially the peroneus brevis, and the invertor or tibial groups, especially the tibialis posticus. They are antagonistic in their function.

Locomotion

In locomotion the foot must also act as a lever. In walking the foot bears weight first at the heel (heel-strike), then shifts it forward to the entire metatarsal region, and finally moves it slightly more to the medial (or tibial) side at the last moment of push-off. In the normal foot the chief walking muscle is that of the calf, the gastrocnemius-soleus group, and for this purpose it is three times stronger than any other muscle in the foot. By raising the heel from the ground it propels the body forward at push-off. When it is paralyzed, there is no adequate muscle transplant to replace its function. It is assisted by the peroneus longus, which acts to depress the first metatarsal head. Though the function of the long toe flexors is also assistive in plantar flexion, actually the main forward thrust in push-off comes from the metatarsal heads, and the metatarsophalangeal joints of the toes are dorsiflexed. Though one would think that push-off could be accomplished only by active muscle contraction, it is a fact that passive resistance of the foot to dorsiflexion will raise the heel from the ground, and thus some leverage will be applied pushing the body forward. This is an important consideration when treatment of the paralytic foot is being considered.

The dorsiflexing muscles, the tibialis anticus, peroneus tertius, extensor hallucis longus and extensor digitorum longus, serve two functions. The first of these is to lift the foot up so that it will clear the ground easily during the swing-through phase of gait, and the second is to check-rein the foot at heel-strike so that the forefoot will not slap down.

Tibial Torsion

Statically speaking, the axes of motion of the ankle and knee joints do not lie in the same plane. There exists an external twist or torsion

in the tibia which causes the plane of the ankle to face outward with relation to the knee. During locomotion this becomes important, since it causes the foot to point slightly outside the line of progression, and thus the weight thrust tends to gravitate more to the medial side of the foot.

ETIOLOGY

There are several types of paralysis which commonly lead to the paralytic foot deformities under discussion, and some confusion can be eliminated if a fairly simple outline is followed.

1. MUSCLE FIBER: Progressive muscular dystrophy
2. PERIPHERAL NERVE
   a. Trauma
   b. Charcot-Marie-Tooth disease
3. CAUDA EQUINA
   a. Meningomyelocele with severe paralysis, including bladder and bowel
   b. Milder congenital abnormalities of the spine and meninges
4. LOWER MOTOR NEURON: Poliomyelitis
5. SPINAL CORD: Friedreich's ataxia

All these lesions are seen in children and produce a flaccid type of paralysis; therefore they can be considered together. Spastic paralysis associated with upper motor neuron lesions and cerebral palsies may also lead to severe foot deformities which superficially resemble those under discussion. Actually, however, there is enough difference in methods of treatment to make simultaneous discussion impossible.

Friedreich's ataxia and the muscular dystrophies are progressive diseases, and for this reason treatment will usually be conservative. Rarely, however, progression is so slow that bony surgical intervention is justified. Obviously, tendon transplantation is never to be considered in either condition.

The most common cause of ankle and foot deformities is poliomyelitis in which paralysis is purely of motor type. Those due to cauda equina and peripheral nerve lesions are often associated with sensory loss, and when using braces, one must be careful to prevent undue pressure against the skin. In the presence of poor sensation, bony healing after operation is often faulty, and therefore special types of procedures may be necessary for these cases.

PATHOLOGIC CONSIDERATIONS

Normal Development

The normal development of foot and ankle depends to a large extent on growth of cartilage centers which are then ossified. During growth, however, the skeletal parts are relatively plastic, and their shapes are

continually molded by the static (gravitational) and dynamic (muscular) forces being applied to them. As long as these forces are evenly balanced, no deformity will develop, but should one muscle or one group of muscles be paralyzed, the unopposed strength of the antagonistic muscle or muscles warps the foot out of shape. The earlier in life the paralysis occurs, the greater the anatomic disturbance is likely to be.

Changes in Paralyzed Muscles

All degrees of muscular paralysis can occur. Since every muscle is innervated by many anterior horn cells, the extent of paralysis is directly proportional to the number of anterior horn cells destroyed or the number of peripheral nerve axons severed. The affected muscle fibers atrophy, and the size and power of the muscle are correspondingly and permanently reduced. The degree of paralysis may be accentuated by nonfatal disease of other anterior horn cells or their axons. Recovery of these is possible, but can be retarded if the muscle is stretched.

Changes in Antagonistic Muscles

The antagonistic muscle, no longer finding itself resisted, pulls the foot in the direction of its action and tends in course of time to shorten. Not only do the muscular fibers contract, but also the connective tissue elements in the muscle hypertrophy.

Ligamentous and Fascial Changes

Once the joints of the foot have been allowed to remain in a deformed position, the ligaments on one side contract while the others become attenuated. These changes also occur in the surrounding fascial structures.

Development of Fixed Deformity

For a time the changes are reversible, but gradually they become more and more fixed. As the limb grows, the shortened structures resist the increase in length, and deformity is aggravated. Eventually, actual structural changes take place in the bones.

Prevention and Treatment

Prevention and treatment of deformity are based on fundamental pathologic observations. *Support* the paralyzed muscle and *stretch* its

antagonist. Preserve joint movement. In the reversible stage, restore muscle balance when possible by tendon transfer. Fixed deformity, on the other hand, must be corrected by section of tight ligamentous and fascial structures or by removal of bony wedges when the skeletal parts are deformed.

TYPES OF DEFORMITIES

Equinus Deformity

When a foot becomes fixed in a plantarflexed position, an equinus deformity exists. When it is severe, the body weight is born entirely

Fig. 54. Fig. 55.

Fig. 54. Equinus with some varus.
Fig. 55. Severe equinovarus deformity. Note that the head of the talus is prominent on the lateral border of the foot (arrow).

on the tips of the metatarsal heads, and the disturbance of gait is typical. The heel does not touch the ground, and the sole of the shoe is badly worn. There are heavy callouses under the metatarsal heads, whereas the plantar skin under the heel is pliable and soft and shows no sign of weight bearing. In the swing-through phase the foot has to be lifted up high (steppage) so that the toes will not strike the ground.

When mild, however, the abnormality results in almost no disturbance of gait whatsoever. The spring or push-off is perfectly normal, but if one looks closely one will see that the sequence of the stance phase of gait is disturbed and that the toe touches before the heel. In these cases examination of the shoe always helps, since the heel shows less wear than normal.

Equinus position results from paralysis of the dorsiflexing muscles, particularly the tibialis anticus. The deforming force is the pull of the

Achilles tendon. In other words, there is an extensor-flexor imbalance in favor of the latter. Occasionally the deformity can occur without obvious muscle weakness, and in these cases it is due to the postural attitude assumed when lying in bed without support of the foot. A board at the foot of the bed when lying supine, and allowing the foot to hang over the lower end of the mattress when prone, will usually prevent its occurrence.

In the absence of heel-cord contracture the same muscle imbalance will cause a *drop foot deformity*, so called because the foot drops into plantar flexion during swing-through, resulting in a typical slapping of the foot at heel-strike.

Calcaneus Deformity

This is the reverse of an equinus deformity. The foot is fixed in a dorsiflexed position, and the patient walks with a *pedestal* or peg-leg

Fig. 56. Calcaneus of moderate degree with mild cavus.

type of gait, bearing all the weight under the heel. There is *no push-off*, no spring to the gait at all. The heel is short and vertical with marked development and callosity in the underlying plantar skin.

The chief cause is loss of power in the triceps surae or gastrocnemius-soleus group. If the plantar flexors of the toes and peroneus longus are intact (and they often are), there is an associated cavus (high arch) deformity, which produces the typical *Chinese bound-foot* appearance.

This is one of the most difficult foot conditions to deal with, since it is really impossible to substitute adequately for the power of the calf muscle. For the same reason, paralysis does not have to be very marked in order for the deformity to be a severe one.

Varus Deformity

A fixed inverted or supinated position of the foot is called a varus deformity. It is best described by thinking of the talus as fixed and then visualizing the rest of the foot as being turned inwards underneath it. Thus the forefoot is adducted, the heel is turned in underneath the ankle, the head of the talus is prominent on the lateral border of the foot, and most of the weight is borne on the fifth metatarsal head and base, with abnormal calluses at these points. Frequently there is a marked increase in the external torsion of the tibia.

Fig. 57. Varus, left foot. Valgus, right foot.

Paralysis of the peroneal muscles is the chief causative factor, with the deforming pull provided by the strong inverting force of the tibialis posticus and anticus. In a varus foot the surface for weight bearing is markedly reduced, leading to pain from abnormal pressure, and the foot is incapable of adapting to rough surfaces. Because of intoeing, the foot does not brace the body well and young children tend to fall frequently.

Valgus Deformity

A valgus deformity of the foot consists in a fixed flatfooted position. It is the reverse of the varus position. The foot is turned outward underneath the talus, the head of which is prominent medially and depressed against the sole. The arch is obliterated and the forefoot is abducted to produce a toed-out appearance Weight is borne chiefly on the medial side of the foot. Unless severe, this deformity is compatible with good function, since the foot is in a good balancing position. One has only to think of the numbers of flatfooted people in the world to realize this fact. However, when severe, it can be just as disabling as the other conditions by causing abnormal pressure under the

first metatarsal head and under the depressed talonavicular articulation.

Valgus is produced by paralysis of the tibialis anticus and posticus and by the deforming pull of the peroneal tendons. Often in the severe cases an associated equinus contracture of the heel is present.

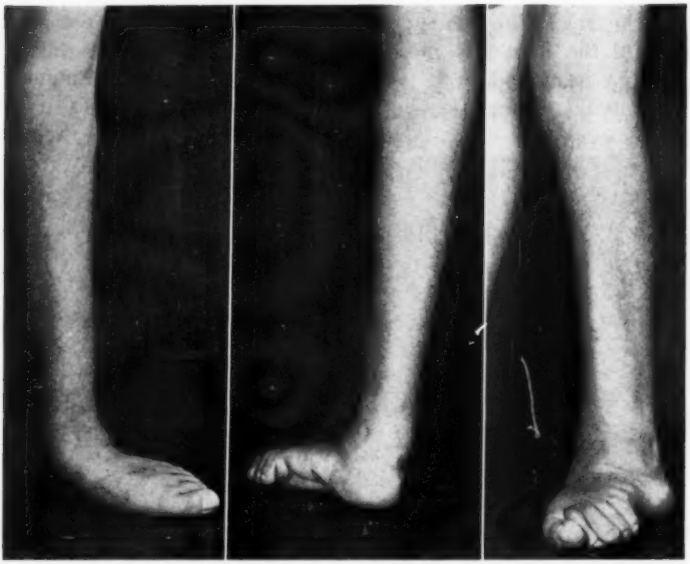

Fig. 58. Severe valgus deformity.

This forces all the weight against the metatarsal heads dorsiflexing the midtarsal joint (talonavicular and calcaneocuboid), causing a *"rocker-bottom" deformity.*

Cavus Deformity

This is a high-arched foot. Weight is borne entirely on the prominent heel and metatarsal heads, and there are usually heavy calluses in these regions. The instep is high, leading to difficulty in shoe fitting. Often there is associated *clawing* of the toes.

Often muscle examination shows no obvious gross muscle weakness, because the underlying paralysis is of the small short muscles of the foot, which are difficult to examine. The deforming muscles are chiefly the peroneus longus and the long toe flexor which depress the metatarsal heads. In addition, the strong plantar ligaments are contracted, a fact which can be readily confirmed by palpating the plantar fascia. The claw toe deformities stem from the pulls of the long toe flexors

and extensors in the absence of the stabilizing actions of the short plantar muscles on the interphalangeal and metatarsophalangeal joints.

Dangle (Flail) Foot

When the muscles of the foot and ankle are totally paralyzed or when nothing but a trace of muscle action remains, a flail or dangle foot results. Usually there is no fixed deformity. However, contracture can be induced by inadequate support of the foot in the presence of prolonged bed rest, or by the use of improperly fitting braces. Especially serious is the latter. Commonly a dangle foot is only part of a flail lower extremity, and in these cases long leg braces are invariably used to provide stability in walking. In making such braces nine bracemakers out of 10 will align the axes of the knee and ankle joints in the same plane. When the brace is put on, the foot is forced into inversion to compensate for the lack of external torsion, which is not only normally present, but often increased in these extremities. The result over the course of time is the production of a fixed varus deformity. This has been seen commonly enough that it should be emphasized over and over again.

Associated Toe Deformities

Deformities of the toes lead to abnormal pressure points with painful callosities and for this reason may interfere markedly with normal gait. There are several characteristic types.

Claw Toe. This contracture is seen in other conditions besides cavus. Sometimes it is difficult to ascertain exactly the causative factors of

Fig. 59. Claw toes with corns overlying great and little toes.

the deformity, and static pressures from abnormal use of the foot often are contributory. There are two elements to the deformity: *(a) cock-up deformity*—a dorsal contracture of the metatarsophalangeal joint; *(b) hammer deformity*—a flexion contracture of the proximal interphalangeal joint. Both are often present at the same time. In addition, there may be a contracture of the distal interphalangeal joint causing a painful callus at the tip of the toe near the nail.

Cocked-up Great Toe. This is usually not a fixed deformity, but a position assumed during the swing-through phase of gait. It is caused by active contraction of the extensor hallucis longus either in trying to

Fig. 60. Dorsal bunion. Note prominence at base of great toe.

overcome an equinus position or in trying to raise the foot when the tibialis anticus is absent. There is usually a callus over the interphalangeal joint, and often the first metatarsal head is prominent in the sole of the foot.

Dorsal Bunion. This is a condition in which the first metatarsal head protrudes dorsally while the great toe becomes fixed in a plantar-flexed position. It is caused by action of the tibialis anticus against an absent or weak peroneus longus, and is aggravated by pull of the flexor hallucis longus with absence of the extensor hallucis longus.

Hallux Valgus. This is the common *bunion* condition of older people, and in paralytic foot cases is the result of valgus deformity. It is usually a statically induced condition, brought on by out-turning of the foot so that at push-off all weight is thrown against the tibial border of the great toe.

Mixed Deformities. All these deformities have been described in their pure forms, but more often than not they are seen in mixed states.

Thus a calcaneus can be associated with either a valgus or varus lateral deformity, and this is also true of an equinus. Furthermore, there are often other paralyses and other deformities elsewhere in the lower extremity which complicate the picture. Often the paralytic foot does not develop normally and is shortened as the result of vascular changes.

TREATMENT

Nonoperative Treatment

Prevention of deformity is one of the most important therapeutic measures. It can be accomplished in several ways:

1. Splinting of the paralyzed foot in neutral position can be accomplished with foot boards, plaster splints and night braces. It is particularly necessary if involvement is severe, and when a strong tendency toward development of deformity has been established.
2. Graduated active exercise of the paralyzed muscles to increase the power of their remaining intact fibers. This is most important in the early phases of paralysis.
3. Stretching of the tightened antagonistic muscles and fascia.
4. Braces are used for one of two purposes: either to stabilize a paralyzed foot and ankle so that they can support body weight, or to prevent development or recurrence of deformity. They should never be used to correct deformity. Other types of supports are also helpful. These include shoe wedgings, foot plates and special shoes.
5. Occasionally, in the presence of severe deformity, wedging plaster cylinders can be used to overcome a contracture so that a brace may then be worn or in preparation for tendon transplantation.

Surgical Correction

Operative intervention is often indicated. This is true even in the absence of symptoms, since correction of deformity is involved, and not relief of pain. These procedures are best done before adult life, since children adjust much more rapidly to the alterations of joint mechanics entailed in doing them. The decision for operation and the best time to do it is often difficult, and to insure the best results these cases should always be supervised by orthopedic surgeons experienced in the problems of paralytic deformities.

Tendon Transfer. In the presence of muscle imbalance it is often wise to shift the tendinous insertion of an intact muscle in order to use it to better advantage. All types of transfers are possible. For example, to replace an absent tibialis anticus, one peroneal tendon should be transferred to the medial border of the foot. Its action can

be enhanced by simultaneous backward shift of the extensor hallucis longus to the first metatarsal neck.

The type and distance of transfer should be planned from analysis of the existing muscle situation. It must be remembered that such

Fig. 61. Fig. 62a.

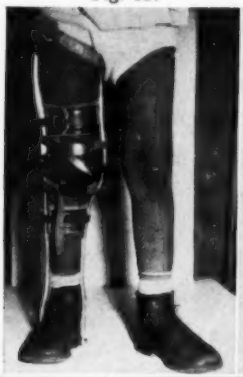

Fig. 62b.

Fig. 62c.

Fig. 62d.

Fig. 61. Long leg brace with ischial ring. Note externally rotated plane of ankle in relation to plane of knee.

Fig. 62. Various types of short leg braces. *a*, Single outside bar with inside T strap and right-angle stop, for control of equinovalgus deformity (as photographed). It can be reversed for varus deformity. *b*, Double upright brace with foot plate and limited ankle motion, shown for flail foot. *c*, Posterior spring steel brace for drop foot. *d*, Steel reinforced molded leather brace for calcaneus.

tendon transplants may work in two ways: they not only *replace* lost tendon function, but often and at the same time *remove* a deforming factor as well. Thus the site to which the tendon is to be transplanted must be chosen only after consideration of these two factors.[9, 10]

Tendons are frequently transferred into the heel, although they can-

not fully restore the function of the triceps surae—that is, provide push-off. The purpose of such transplants is to stimulate more normal growth of the heel by the molding action of muscular contraction.

Tendon transfers should never be done until after deformity has been corrected, and a strong nonparalyzed muscle, if available, should always be used. Surprisingly enough, re-education of the transplanted muscle is seldom if ever a problem after such operations.

Tenotomy and Fasciotomy. Occasionally a tight and contracted tendon or fascial structure will have to be severed to allow correction of

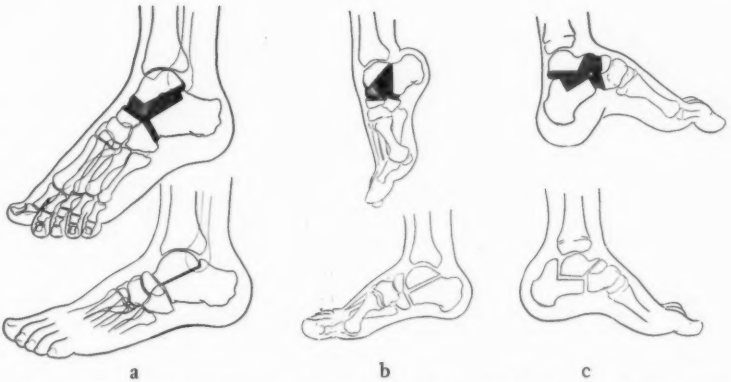

Fig. 63. Triple arthrodesis. *a*, Bone to be removed for stabilization in a foot without much deformity. *b*, Bone to be removed for correction of equinus (Lambrinudi's operation). *c*, Bone to be removed for correction of calcaneus (reverse Lambrinudi).

deformity. A common example is heel-cord lengthening to overcome equinus. These procedures, however, are practiced as infrequently as possible because they sacrifice valuable muscle power.

Osteotomy. Bony deformity can be corrected by osteotomy; for example, to overcome increased external tibial torsion.

Bone Blocks. Also, certain postural attitudes of the foot can sometimes be overcome by creating bone blocks. Such procedures are particularly useful in preventing excessive valgus and drop foot in very young children when more extensive procedures are not advisable.[3, 6, 13]

Triple Arthrodesis. Once bony deformity is established, it must be corrected by removal of suitable bony wedges from the tarsal region. The wedges are varied according to the type of deformity. In so doing the function of the subtalar, calcaneocuboid and talonavicular joints is sacrificed, resulting in improved stability. The function of such feet is usually excellent even in the presence of marked muscle weakness. One should always correct the foot into a slight valgus position in order to insure best stability. Because the procedure removes cartilage

and interferes with bone growth, triple arthrodesis should not be done until the foot is fully matured (ages 10 to 12).[1, 2, 4, 5, 7, 8, 12]

Panarthrodesis. This is an operation in which ankle fusion is combined with triple arthrodesis. It is often the procedure of choice in the presence of severe calcaneus or equinus deformities. It provides a strong foot with some passive push-off as long as the foot is fused in a position of moderate equinus.

Astragalectomy and Posterior Displacement of the Foot. Removal of the talus is a worth-while procedure in paralytic calcaneus when there is associated loss of sensation, since it avoids the danger of nonunion which sometimes complicates triple arthrodesis in these cases. It is also occasionally a useful procedure in poliomyelitis cases when calcaneus is severe and the child too young for triple arthrodesis. It results in greater stability of the foot by placing the ankle closer to the ground and by destroying subtalar movement.[11, 14]

Correction of Claw Toes. This can be accomplished by a variety of relatively simple procedures. *Proximal phalangectomy* is one of the most useful of these operations. Sometimes, however, *wedge resection and fusion of the proximal interphalangeal joint* is to be preferred. For the tender corns that develop at the tips of the toes *terminal Syme amputations* which remove the toenails are successful.

SUMMARY

Foot deformities developing from flaccid paralysis are due to muscle imbalance and can often be prevented by adequate conservative treatment. Several types of deformity are recognized and are produced by typical patterns of imbalance. All of them result in weakening of the foot and impairment of its function.

In the presence of deformity, function can be improved by a variety of operative procedures, some of them done on soft tissues alone and others on the bony structure.

Proper supervision is the most essential part of conservative or surgical management. It should begin from the moment paralysis is recognized and continue until the foot and ankle are fully matured.

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SHOES FOR CHILDREN

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The subject of children's shoes is worthy of discussion because it has long been neglected by the medical profession. This lack of interest by the physicians at large has allowed the shoe manufacturers to gain the position of not only advising the laity, but also influencing the medical profession in regard to the "proper shoes" for children. However, it is improper to subject these little patients to unqualified nonprofessional groups, because the mother who inquires as to the proper shoes for her child is entitled to sound advice, based upon examination and diagnosis, in the same manner that recommendations are given for any other medical problem. The fact that a shoe has been advertised as "proper" should not influence the judgment of the physician.

Inasmuch as it is the pediatrician who is called upon to examine the feet at birth and again upon walking, it is necessary for him to be cognizant of the ordinary foot problems in children. With this information the pediatrician will be able to prescribe the proper shoe with the proper corrections and will not be dependent upon the judgment of the shoe salesman. In this way the patient will receive professional treatment as opposed to pressure salesmanship.

It is not the intent of this paper to discuss either the pattern of each abnormal foot condition or the type of footwear necessary for each condition. Such information has been published elsewhere.[2, 3] However, a word can be said about normal feet, because, should the structure of the normal foot be known, variations from the normal can be distinguished easily.

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SHOES FOR NORMAL FEET

In Figure 64 a normal foot presents a good longitudinal arch without the presence of pronation. The forefoot is well aligned on the rearfoot, and the toes are well spaced and straight, and lie flat. The ankles do not bulge internally or externally, and the foot as a whole is straight forward and properly aligned on the leg.

Shoes which are considered proper for children should be of the blucher type and lace across a tongue. They should be well made of solid leather with thick, inflexible soles. In general, they should be

Fig. 64. Normal feet. Note the good longitudinal arches without the presence of pronation.

oxfords, inasmuch as high shoes offer no additional support and are of no value except for protection against the elements. Booties, and artificial leather, rubber sole and soft sole shoes never should be used. With proper shoes the desired corrections may be obtained by first diagnosing the patient's condition, and then ordering by prescription the appliances which are indicated. In this way the patient receives specific treatment for the specific defect.

ORTHOPEDIC SHOES

The term "orthopedic shoe" should be discarded because it implies that a shoe has been specially constructed for the child's foot, and yet it is patent that no single pair of shoes can be correctly constructed to take care of all the defects which children may show. It implies further that the manufacturer is treating a patient who has never been seen. In such instances the manufacturer builds into the shoes all the appliances known to the orthopedist, in a more or less "shotgun pre-

scription," to cover all the conditions which might exist. In consequence, the patient may be damaged by certain appliances while being aided by other corrections.

Figure 65 shows on the left a pair of orthopedic shoes with built-in appliances and swung-in toes, while on the right is a pair of normal shoes. Figure 66 is a typical example of the unfortunate use of so-called orthopedic shoes. In this particular instance the feet were entirely

Fig. 65. Shoes. Note so-called orthopedic shoes on the left, compared to normal shoes on the right.

Fig. 66. Shoes. Note shoes with swung-in toes on the left, which produced callosities on fifth toes of normal feet in the center, relieved by straight shoes on the right.

normal, as can be seen in the center, but a standard make of orthopedic shoes was prescribed for the patient. The swung-in toes of the shoes on the left obviously could not fit the straight normal feet of the child. The result was thick callosities on the lateral aspect of each fifth toe, which by close examination can be seen. After the removal of the orthopedic shoes, and the substitution of the straight normal shoes on the right, the callosities disappeared rapidly and the patient's complaints ceased.

WEDGING OF SHOES

Much confusion exists as to the manner in which shoes should be wedged for purposes of correction of the various deformities of the

feet. Wedges are of value only when applied to solid leather shoes with thick soles. They should be placed outside and not inside the shoes, and should be inserted between the soles and within the substance of the heels so that they are not subject to wear and tear. Their thickness is thus maintained, and, after being properly and accurately placed by an orthopedic shoemaker, the shoes may be half soled and the heels may be built up by an ordinary shoe repair shop. The wedges should vary in width from ⅛ to 3/16 inch, depending upon the age of the child and the degree of deformity.

Figure 67 demonstrates the common types of wedges. Figure 67, A, represents an inner heel wedge used for pronated feet; *B* demonstrates

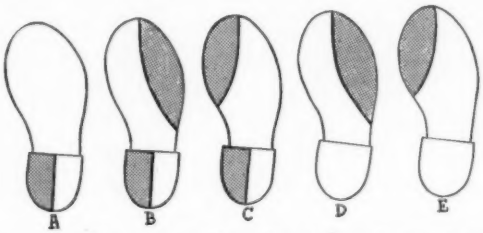

Fig. 67. Shoe wedges. Note proper position for, A, inner heel wedge; *B*, inner heel and outer sole wedges; *C*, inner heel and inner sole wedges; *D*, outer sole wedge; and, *E*, inner sole wedge.

inner heel and outer sole wedges used for weak feet; *C* represents inner heel and inner sole wedges necessary for severe relaxation of the longitudinal arches in talipes calcaneovalgus (inasmuch as almost all shoes for children up to the age of six years, or size 12, are made without heels, the wedge may be placed throughout the medial border of the shoe); *D* demonstrates an outer sole wedge used for the postural type of metatarsus varus; and *E* represents an inner sole wedge used for the postural type of external rotation deformity.

FITTING OF SHOES

In regard to shoe fitting, mention should be made of the danger which is inherent in the use of the fluoroscope, by certain retail stores, in the fitting of children's shoes.[1] The hazards are uncontrolled length of exposures as the mother travels from store to store, continues to fit shoes after shoes, plus the mere curiosity on the part of the customer. To date injuries have not been reported, but this does not imply that skin damage or epiphysial distortion may not have occurred, because sufficient time may not have elapsed for such injuries to become apparent or because such injuries may have occurred and been attributed

to other causes. Shoes always have been and may still be fitted by the ordinary methods of measurement and observation, particularly when there is nothing more to do than to have the shoe one thumb-breadth longer and one little finger-width wider than the foot. It appears that fluoroscopy for shoe fitting may be used more on an advertising basis than on a scientific basis.

SUMMARY

1. Normal feet and proper shoes are defined.
2. The disadvantages of orthopedic shoes are discussed.
3. The correct wedging of shoes is illustrated.
4. The fitting of shoes is described.

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POLIOMYELITIS

The Demands of Continuing Care

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During 1954 there were 38,741 new cases of poliomyelitis reported in the United States. Because of more detailed and frequent muscle testing, it is now realized that few patients escape with truly nonparalytic disease.[9] Children continue to predominate in the yearly incidence. As growth progresses, the consequence of involvement can become serious, and maximum crippling may not develop for many years. This fact is most distressingly represented by a full-blown structural scoliosis six years after the acute attack (Fig. 68). Conversely, an infant with considerable involvement can be brought along to a successful adult life by the continued application of a sound treatment program.[8]

In this era of speed and space it is only natural to seek short cuts in treatment. The care of a patient with even minor weakness requires time. The more severe the paralysis and the younger the patient, the longer must one persevere with treatment. Some things cannot be hurried, and the recovery process in poliomyelitis has a "deliberate speed" which must be honored. To prolong a restricted conservative program is lamentable, but, particularly in the first year after onset, this is wiser than trying to force recovery. Certain considerations of the disease process will clarify this point.

PECULIARITIES OF THE DISEASE PROCESS

The Nature of the Patient

Let us start with the host who becomes infected with a poliomyelitis virus. There no longer appears to be a specific diathesis as far as somatotypes are concerned. At least those with severe paralysis may be brunette or blond, lanky or chunky. It is still generally true, however, that the chronically ill and undernourished rarely become badly in-

volved. Usually the patient is of sturdy stock, a fact noted originally by Jacob Heine more than a century ago.

Certain individual characteristics are important in relation to the total disability from a given amount of paralysis. For instance, an obese, lackadaisical subject will generally have a much greater problem

Fig. 68. Sitting roentgenogram of a 12 year old girl taken 6 years after onset of poliomyelitis. The scoliosis had changed little for 4 years and then progressed rapidly.

than a slender "live wire" with similar losses of muscular power. Some children are found to be unusually loose-jointed—the type with severe flatfeet and knock knees at three to five years of age. Serious problems of joint stability occur with weakness of crucial muscles around the hip and knee. Youngsters with ligamentous relaxation are more prone to paralytic dislocation of the hip and to severe back-knee deformity, and if obesity is superadded, the insult can be tremendous. We have all been impressed by certain persons who lack coordination and agility. If such a "muscle moron" is afflicted by flail lower extremities, a wheel

chair may be the only practical means of getting about. Another person similarly involved, but with athletic timing and skill, may be on his feet and walking successfully with crutches and braces. All these factors, useful or harmful, must be borne in mind as treatment continues and a functional prognosis begins to take shape.

Correlation of Pathologic and Clinical Findings

Our next concern is the interaction of host and infecting virus. Necropsy studies on animals and man have indicated more widespread invasion of the nervous system than would have been suspected by evident signs and symptoms. Specifically, a critical number of cells must be knocked out of function before ordinary methods of examination reveal involvement. Bodian[2] showed, for example, that in the rhesus monkey the anterior horn motoneuron pool has considerable reserve. No paralysis could be demonstrated, but by postmortem study of sections as many as one third of the cells were found to have been infected by the virus. This observation explains why minor weakness in some muscles is detected only by repeated, detailed examinations, and hence the rarity of completely nonparalytic infection.

Much is now known of the pathologic histology of severe damage to the central nervous system with extreme paralysis. The pathologic physiology of disturbed function in the central nervous system has almost defied investigation, but we can assume that there are widespread repercussions in neuromuscular control. Patients without gross paralysis may be irritable and lack endurance for many months. Parents often volunteer that a child with only minor involvement is more easily upset and subject to emotional outbursts when tired. Whether this represents disturbed physiology in the central nervous system or is an alteration in behavior induced by anxiety and restrictions cannot now be verified. The important fact is that such occur and in some hypersensitive children may become a serious problem in adolescent and adult life.

By careful study it has been shown repeatedly that the recovery of weakened neuromuscular units is rapid in the first six months after onset.[3] In fact, the maximum strength is usually regained within 18 months. Endurance, however, may require much longer to reach its peak. It has been found that overuse of a weakened muscle can delay or even prevent its expected good recovery. For example, with involvement of the calf group, too much walking and activity which requires raising the body weight can cause serious damage and even permanently impair function. For many months after the initial involvement patients may have muscular cramps and aches, especially at night after considerable activity. This is apparently related to metabolic disturbances

from fatigue. Some believe,[1] however, that during convalescence the usual sensory appreciation of fatigue is altered and the natural safety-check against overdoing is not reliable. At any rate, whereas the obvious recovery of a paretic muscle as judged by a momentary hand test may be complete within a matter of months, the restoration of the body from an attack of poliomyelitis may well require years.

WHAT IS ADEQUATE TREATMENT, AND HOW CAN IT BE ASSURED?

The public today has been told much about poliomyelitis—its cause and treatment. Consequently even young children have become poliomyelitis-conscious, and their parents come to expect near-miracles. The sobering fact remains that there is still no specific cure, and the only real solution lies in prevention Faced, then, with immutable paralysis, most of us do not want to believe it. After a while both patient and parents are apt to look afield from conventional medical care when the desired cure appears impossible. It is the great responsibility of the physician to realize what an important role he must play in a very personal way once he undertakes to treat poliomyelitis. He must gain and hold the absolute confidence and cooperation of the patient and family. His therapeutic wares are apt to lose their lustre as time passes, and he must be wise in his counsel about consultants. Since the problems require considerable breadth of medical knowledge, including the specialties of pediatrics, neurology, physiatrics and orthopedics, he must honestly determine his own forte. The more comprehensive his ken of the treatment program as a whole, the better can the phases be correlated so that his contribution will be the greatest.

The Aims of Treatment

The goal of treatment can be stated simply: to attain the maximum possible functional recovery of the whole patient. The steps toward this ultimate goal are also familiar: namely, (1) the facilitation of the natural recovery of weakened neuromuscular units by (2) careful physical and surgical measures which produce coordination and muscle re-education and increase the strength and functional ability. In turn, these will be interfered with by the secondary changes in the musculoskeletal system if contractures and deformities are permitted to occur and progress. These general aims are often repeated and accepted to the point at which the underlying principles and the details of their application may be overlooked.

Since poliomyelitis remains predominantly a disease of childhood, the end result must not be reckoned in terms of five or 10 years, but should be judged by the probable condition in the fourth and fifth

decades or later. It is then that the person is reaching the peak of worldly performance, and the family responsibilities are great. The appearance of degenerative changes may bring to the surface symptoms which, dormant until then, can produce progressive crippling.

An unforgettable example is shown in Figure 69. The patient is 41 years old, had acute poliomyelitis at age three, and discarded the left long leg brace at age 13. At age 40 (27 years later) he first began to have pain in the left knee from strain. The disability is becoming greater, although the deformity has not changed.

Fig. 69. Severe recurvatum of the unbraced left knee present for 27 years. No pain occurred until age 40. Note that the right knee is kept out of deformity by the brace and is asymptomatic.

During the convalescent period of the first year or two the physician must keep in mind the probable condition of the patient not only at the end of growth, but also as an adult. One should never see how much can be got away with during the early years if a more reasonable and cautious program can produce worthy and continued results. Let us be specific.

Phases of Treatment

In dealing with poliomyelitis one is constantly impressed that "a child is not a little man." The year old baby and the five year old, the adolescent and the adult, each must be dealt with individually even though the same general principles of treatment apply to all. Usually

it is best to give the young child a longer period of protected activity to allow for maximum recovery. The adult's prognosis may become evident within a few months, and the successive phases of muscle re-education, standing and walking can be completed in a much shorter time. The adolescent is more conscious of appearance and conformity to the group standards and rebels against such equipment as high top shoes or canes. The five year old must keep up in physical activity with his playmates no matter how badly he limps, and voluntary gait and posture control are practically too much to expect. The year old can be regimented remarkably well in company with others in a similar plight in the ward environment.

Take a common principle: prevent deformity by avoiding faulty alignment of the bodily segments. During the early weeks of sensitivity and painful spasm one may have to accept undesirable positions to obtain comfort and rest. As soon as possible, however, the patient in bed should be kept out of deformity. To prevent malposition of the lower extremities the infant may require a special frame and restraints; the child, removable bivalved long leg casts; and the adult, a foot board. Get the patient into the "position game" as early and completely as possible. Look at the youngster with a tight low back and weak abdominals. In the supine attitude the alignment looks good, but come back a bit later. Here's the patient now lying prone, propped up on the elbows to see what's going on, and what a hollow back is being produced! Get the nurses into the picture, since they are essential for this part of the program. If the patient is at home, the family must understand these disciplines and carry them out effectively.

The time comes to sit up. What then? The principle still holds: avoid habitual malposition. If this can be done for 30 minutes, then one should allow only half an hour sitting up. See what happens when fatigue occurs. Many times the legs will be drawn up into the frog-leg attitude, so that later on the spine becomes curved as well as hollow-backed. Don't let this go on. It may require shorter sitting time or the use of a back rest or a corset. Use what is necessary, and increase activity only within safe limits. Don't be deceived by enthusiastic demands. If bad posture patterns appear, they must be corrected by every reasonable means. It is better to defer a more strenuous activity than to allow too much freedom early and have to curtail later. Think of the effect on morale.

Still the child feels good and is "rarin' to go," but must be restricted. Too often parental pity and conscience enter here. Poor child, they think, let's give him something to make up for this sad situation; something like candy, cake and ice cream; a party every day. This is the opening wedge for obesity, which can become the obstacle that hamstrings future walking and function. How much better to put the mind

and hands to work and supply books, crayons and model airplane kits instead of sweets and sympathy. Start a club on the ward or in the neighborhood. Encourage; enforce discipline wisely but well.

To Brace or not to Brace

At long last the glorious day arrives when standing can be resumed. At first weight bearing may be started on a tilt table or in a pool and later on dry land. Faltering steps are taken, and crutches with manual guidance provide security. It is discovered that the full body weight can be supported on one extremity without a brace, but the other buckles. What then?

Let us go back a decade to when the Kenny enthusiasts were so vociferous. Among other loud pronouncements, braces were condemned as instruments of torture. I resent this. When indicated, proper braces are instruments of liberation! Here is the evidence: We are striving for the "maximum possible functional recovery of the whole patient." Observe a patient standing and walking. Ultimately this means not in your office, but "under fire," at home, at school, on the playground. If the *usual* stance is one of deformity, this must be avoided. If the gait is faulty and the trunk lurches badly to one side, this must be corrected. One cannot continue to restrict activity in a growing child without adversely affecting the behavioral development. Can the loose-jointed lad with a poor quadriceps have stability only by locking in the back-knee position? If so, he should have a brace to provide support without deformity (see Fig. 69). If a lass has severe paralysis of the hip abductors and extensors, a crutch or cane in the opposite hand is a sound solution to prevent the unsightly, spine-twisting lurch. With appropriate apparatus, much activity is safe and sound.

The goal is not to go without a brace, but to continue *over the years* to do what the patient needs and deserves to do, practically and with the least mechanical insult to the body. The younger the patient, the more one must control by braces and apparatus for two reasons: First, voluntary regulation of gait in a "live wire" youngster is out of the question. Second, the long period of growth makes potential disasters of deformity from even slight variations from normal. If control is satisfactory by late adolescence, braces can often be shed with or without surgery. When the adult honestly finds that he can safely discard apparatus, he should do so,[7] provided the anticipated demands of future life will not be greater. At times it is best to use a brace or cane for certain strenuous or hazardous activities, and go without it part of the time when the situation is easier. Analyze the patient's daily needs, and if apparatus permits more nearly normal function, use it.

Above all, the physician must take a positive attitude and be dynamic

in those measures which have proved worth while over the years. Remember, *more crippling results from the insult of deformity than from paralysis alone*. When conscience dictates that by getting away with mechanical murder in childhood future fixed deformity will later cause grief, have courage and avoid it if at all reasonable.

Some Problems of Returning to a "Normal" Life

Usually about four to eight months after onset of paralysis several things happen. The patient (and family) become aware of the amount of permanent involvement. The benefits of physical therapy are less obvious, and the daily rigorous routines of limited time up and walking begin to get on the collective nerves. Most patients who have been hospitalized for convalescent care are able then to return home. If they have remained at home throughout these early months, they are ready for more strenuous activity, but not unlimited in type and degree.

So much damage can result even within a few months that proper follow-up care must be practically guaranteed. The crux of this, of course, is the family. If they are relatively intelligent and understanding and reasonably cooperative, the future is much brighter. Take them and the patient into your confidence and outline the essentials of everyday living. Make sure that the home routines are clear and within their reach of mentality and performance. It is ridiculous to ask that they spend an hour or so twice a day doing specific stretching procedures and exercises. In the first place, such an amount is likely to cause harm by fatigue rather than to produce benefits. Also, neither patient nor parent can carry this out properly for that length of time.

One cannot expect the unreasonable if braces, corsets and other types of apparatus prescribed are too complicated. One must explain and insist on the simplest essential schedule and underscore definite "dos and don'ts." Obviously this cannot be done only by a mimeographed sheet of instructions. Here is another time when the physician's personal rapport is so important. He must demonstrate his unfailing interest in the patient's future progress and problems and assure this by arranging for regular check-up examinations. At each visit the needs are re-evaluated and activities increased or modified.

The physician must know enough about the home environment and economic situation of the family to anticipate daily needs. It is too seldom possible to have a visiting nurse or physical therapist come regularly to the home. Often the mother can be trained to do the necessary nursing and physical therapy, and she should get a maximum response because she has one patient whose outcome means success or failure to her. But if she is too ignorant or has too many other

family and household responsibilities, she may be unable to take on the job.

A crippled children's school may be the next best answer for a youngster with considerable involvement. This provides supervised care and competition geared to the badly paralyzed patient's level. If, however, the patient commutes as a day pupil, the benefits may be entirely counteracted by other factors. For instance, if the child must leave home at 7 A.M. and return at supper time, the distances and consequential fatigue may be prohibitive. A home teacher, practical nurse or conscientious attendant may be required to attain the goals at this stage.

Let us consider the school-age child with paralysis of one lower limb and some weakness of the trunk, who has been cared for entirely at home. The problems differ in several important respects. During early convalescence obesity from overeating is more apt to occur. When he can be allowed up with, say, a long leg brace, crutches and corset, he may find himself surrounded by unrestricted children both at home and at school. Unlike the very severe case, he is almost an equal in competitive activity, yet he is held down to certain limits of type and time. He is obviously a cripple, and often children are likely to take advantage of this by verbal and physical abuse. He cannot run as fast, and with crutches his hands are tied. Soon he learns to go without crutches or brace, or both. His limp then becomes a real problem.

Here again individual analysis is essential. At first a half day at school and a rest period after lunch may be enough to allow usual activity the rest of the day. With great drive and much energy he may still be difficult and irascible, especially when tired (many are). Often tricycle or bicycle riding allows for enough exercise without the dangers of less controllable walking and running. The manipulation of the play environment may help. Group activities of a manual type which are appealing at that age level should be tried. Whenever possible, swimming is an excellent activity if the hazards of respiratory infections are controlled. If the child gets too strenuous, then at least a part of each day must be devoted to posture and gait practice lest the good habits be destroyed. It takes a nice balance between too much restriction leading to self-pity or reactive aggression and too much activity leading to physical harm and more crippling from consequential deformity.

Indications for Operation

Many patients with paralysis are eligible for improvement by surgery. The timing and indications for various procedures are tremendously important. It has been said again and again that no operations are in

order until at least two years after paralysis has occurred. There are several important exceptions. In general, if contractures of the feet and legs are becoming severe enough to threaten bony deformity, surgical correction may be indicated within the first year. Contractures of the plantar fascia, heel cord and iliotibial tract are particularly apt to need surgery.[6] Frequent consultations with an experienced surgeon should be arranged so that the true progress can be judged by personal examination and the proper time for intervention selected by him.

Operations to correct bony deformity or to provide joint stability are usually not necessary until two or more years after paralysis. This generality applies also to procedures which improve function such as tendon transference in the extremities. The age of the patient is an important consideration. Joint stabilizations of the foot are not routinely advised until the age of nine or older. Even then, if paralysis around the knee and hip makes continued use of a brace advisable until growth is nearly complete, foot surgery may be deferred unless deformity becomes too severe.

The hand and upper extremity are seen more often for surgical rehabilitation than in past years. More patients now need it, and improved techniques have been devised. Usually few operations are indicated before the teens, and these are to combat progressive deformity which cannot be controlled by physical therapy and splinting. Elective tendon transfers to replace lost function are most gratifying.[5] As a rule, after surgery hand splints and many types of assistive apparatus can be eliminated. At times a pathetically paralyzed extremity can be improved dramatically. The hand may hold the key to the essentials of daily function, and every possible physical and surgical improvement should be provided.

Scoliosis

This problem demands the undivided attention of all who treat poliomyelitis.[4] From the early months one must check and recheck the status of the spine by clinical and roentgen examinations. A back may have a minimal curvature for several years and during rapid growth and greater activity abruptly become severely deformed (Fig. 68). Too often the examination is too cursory to elicit important findings and, when such are discovered, too little is done to control serious progression.

Generally the more severe and the more asymmetrical the paralysis, the worse the scoliosis will become. Essential concepts in treatment emphasize certain important principles. First, time is of the essence, and every patient, even nonparalytic, should be examined regularly until growth is complete. Second, the spine is usually straighter in recumbency than in the upright position. Third, by reasonable conservative

measures the spine should be kept as straight as possible for as many of the 24 hours each day as can be done. Fourth, when muscle power permits, posture control (avoiding slumping and potbelly) in sitting and standing must become a natural habit. Fifth, when the trunk is too weak, additional support (corset or jacket) must be used, but mainly as a safety check to prevent the worst deforming attitude from occurring. Sixth, stretching and strengthening of the trunk are important as a means to an end, but not to replace proper postural control or extra support. Seventh, passive improvement of a curvature is obtained by apparatus with distraction and counterpressure, and these are most effective in recumbency; correct when flat and hold while erect. For every hour that the spine is only held by support in the upright position, there should be an hour of better alignment while lying down. Finally, if definite progression occurs despite reasonable nonoperative treatment, spinal fusion should be done before irreversible bony deformity develops. Watch and treat, not simply watch.

CONCLUSIONS

As the encouraging reports of successful vaccination increase, there will be a tendency to underemphasize the thousands of patients who still need continued follow-up care. The problems of poliomyelitis are amenable to satisfactory solutions by the application of sound orthopedic principles, conscientiously and carefully over a period of many years. A team-of-specialists approach is required, but the physician must guide the patient individually and call upon specialist consultants when needed. There are still no drugs to restore paralyzed muscles and none to prevent crippling from deformity which can cause progressive disability even after growth has ceased. If a sound long-range treatment program is followed sensibly, there are few patients who will end up as badly disabled. It is the physician's personal responsibility to see that the patient receives this essential care, and he can then share in the joy of the adult who has conquered poliomyelitis.[10]

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CHILDHOOD LIMP

Diagnosis and Treatment

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Human locomotion is a complex phenomenon. Saunders, Inman and Eberhart[5] in their careful analysis of normal gait have described six major determinants. These are pelvic rotation, pelvic tilt, knee and hip flexion, knee and ankle interaction, and lateral pelvic displacement. The normal human being, therefore, progresses through space by an integration of all these factors.

Limp is caused by failure of one or more of the normal factors to function because of disease. Of the many signs of affections of the skeletal systems in the child, possibly none is more distressing to parents and to physicians than limp. We are so accustomed to the normal rhythm of gait that any variation is almost immediately noticed by both laymen and physicians alike. A diagnosis is not usually made on the basis of a single sign alone. However, some signs are so striking and significant that they immediately suggest a specific disease. Limp promptly implicates the lower extremities as the site of possible disease. Because it is a sign common to many affections of the skeleton and its associated structures, limp merits particular study and careful consideration.

LIMP DUE TO STATIC DISTURBANCES

A static disturbance is one which is due to abnormal deflection of body weight. The abnormal deflection may occur in either the anteroposterior or lateral plane. The deflection may be due to a combination of these factors producing a torsion strain. A child during periods of rapid growth or during convalescence from a serious illness experiences a temporary diminution in muscular and ligamentous support. In either of these circumstances he may have pain about a weight-bearing joint which will produce limp.

Chronic Strain of Internal Collateral Ligament of Knee

A good example of limp due to static disturbance is chronic strain of the internal collateral ligament of the knee secondary to relaxation of the long arch of the foot. In the young child relaxation of the long arch of the foot is often associated with a mild knock knee deformity. Prolonged weight bearing on uncorrected relaxed flat feet causes stretching of the internal collateral ligament of the knee. The knee is a hinged joint and, for all practical purposes, moves only in one plane. Weight bearing on a foot with a relaxed long arch produces a twist or torsion strain upon the knee with each step. The internal collateral ligament eventually stretches and swells. The structure is highly sensitive and becomes very painful. The child develops an antalgic type of limp in which the weight is shifted quickly from the painful limb to the normal side, thereby disturbing normal forward progression of the body.

The *treatment* is simple. It consists in correction of the relaxation of the arch of the foot so that the body weight is shifted to the outer side of the foot and strain is thereby relieved from the inner aspect of the knee. In the young child this can be accomplished by wedging the inside of heel and sole. In the child over five an arch support is required.

Flatfoot

Flatfoot in the child from two to four is usually noticed by parents as a deformity rather than as a condition causing actual disability. The internal malleolus becomes prominent, and the the child walks with the foot everted. Although the gait is somewhat unusual because of the marked eversion of the foot, it cannot actually be described as limp. However, in older children from eight to 12 the condition occasionally becomes painful, and, if so, lameness develops.

The so-called *rigid* flatfoot or *spastic* flatfoot in the child is a relatively uncommon condition. The cause remains somewhat obscure, but recent articles by Harris of Canada indicate that congenital abnormalities in the foot, specifically bony ridges between the talus and calcaneus or calcaneus and navicular, are contributing causes. The foot is held in eversion, owing to spasm in the peroneal musculature. The child walks with the foot everted through the entire progression of gait, and the lower extremities are often externally rotated. As a rule the foot becomes symptomatic early, and the limp is again of the antalgic type.

In the young child *treatment* is difficult. Arch supports are of little value. After the foot has attained most of its growth arthrodesis of the calcaneocuboid, talonavicular and subtalar joints will produce relief and provide a satisfactory weight-bearing foot.

Discrepancies in Leg Length

Abnormal deflection of body weight in the lateral plane is most frequently due to discrepancies in leg length. Minor variations in leg length are often found in the growing child and may measure from ⅛ to ½ inch. Discrepancies of this degree represent variation in growth which is temporary for the most part. The child at three years of age may have ⅜ inch difference in leg length and at six will have legs of equal length. Discrepancies in length greater than ¼ inch are associated with mild limp. The limp is painless, and as the child progresses forward the pelvis and shoulder on the short side drop slightly.

Correction of the abnormality requires equalization of leg length by a suitable heel lift. Leg length discrepancies of an extreme degree, associated with chronic poliomyelitis or severe growth disturbance, are best treated by epiphysial arrest in the longer limb.

Directional Variations in Growth

Directional variations in growth, such as knock knee or bowleg, will occasionally produce an antalgic limp due to pain in the knee or ankle. In both these deformities strain is placed upon hinged joints secondary to variations in their normal plane of function. Knock knee or bowleg may be either developmental or rachitic. Severe bowleg is often caused by internal torsion of the tibia. In order to walk forward without tripping over the toes, the child widens his stance, and the appearance caused thereby is one of marked bowleg.

The *correction* of the limp lies in correction of the directional skeletal abnormality. In the young child nonrachitic directional variations in growth can usually be corrected by shoe wedges. A $\frac{3}{16}$-inch inner heel and sole wedge in a high shoe, if persevered with over a period of several years, will correct the deformity. Occasionally osteotomy is needed, particularly in rachitic children.

Intoeing and Out-Toeing

Intoeing and out-toeing are frequent abnormalities of gait in the young child. While not actually considered limp, they merit discussion. The majority of young infants manifest tibial torsion, which is specifically a twist of the tibia resulting in an inward rotation of the foot and ankle. Tibial torsion noticed at birth is secondary to intrauterine packing in which the thighs are flexed on the trunk, the knees flexed on the thighs, and the feet turned inward in such a position that torsion of the tibia readily occurs. The torsion often reverts spontaneously, but sometimes requires particular treatment. It is noticeable to parents when the child begins to walk as he progresses forward with both feet turned inward. Careful examination with both knee caps pointing up-

ward demonstrates an inward torsion of the tibia so that the knee and ankle do not lie in the same plane. Intoeing, as a rule, is not painful; however, if uncorrected, it will occasionally produce pain in the knee or ankle due to persistent strain on hinge joints. Persistent intoeing, unassociated with internal tibial torsion, is due to anteversion of the necks of the femurs. No treatment that is conservative is of much value. Most of these children improve with growth. Rarely, derotation osteotomy of the femur is required.

Correction of tibial torsion is a simple matter. The child should be fitted with high-topped, stiff-soled shoes with a $\frac{3}{16}$-inch inner heel and outer sole wedge. In addition, he should be fitted with a night splint which consists of a metal bar attached to a pair of shoes. The shoes are placed in external rotation, which is increased gradually as tolerated. The torsion can be corrected entirely if this treatment is persisted in over a period of many months. Children who are face sleepers after the stage of infancy may develop a most persistent tibial torsion because they lie with both legs in internal rotation during sleeping hours. The night splint is particularly helpful in correcting torsion in these children.

Out-toeing commonly accompanies extreme pronation of the long arches of the feet. Sometimes it is due to external tibial torsion, a developmental variant which is seen much less frequently than internal torsion. External tibial torsion also occurs in chronic poliomyelitis as a result of contracture of the tensor fasciae latae and weakness of thigh musculature.

Suitable long arch supports, reverse Denis Browne night splints and occasionally osteotomy of the tibia constitute the therapy of out-toeing.

THE LIMP OF TRAUMA

All children fall frequently, but, owing to the elasticity of the supporting structures of the skeleton and to the malleability of the bones, they sustain few serious injuries from minor trauma. *Sprains* occur relatively infrequently from a single injury. However, sprains do occur in the foot and ankle of the young child which are manifested simply by an antalgic type of limp. The child will cry when the injured extremity is examined and will refuse to bear weight on the affected limb. Symptoms subside spontaneously within a few days or sometimes within a few hours. The child will control his own activities and will not put full weight upon the extremity until the symptoms have subsided.

Overuse of joints, particularly in the adolescent child, can produce limp. With the present emphasis on muscular development and body building inflicted upon the adolescent child in the schools of today it is not unusual to examine male children who complain of intense pain in the lower extremities after excessive exercise. These children have

cramps in their legs at night, and during the day will have a mild limp in one or both lower extremities with complaints of pain about the hip, knee or ankle after prolonged walking or vigorous exercise. Careful examination will demonstrate some restriction of motion in the lumbar spine, tightness of the hamstring muscles in both legs, and slight discomfort in the knee and ankle at the extremes of motion. The diagnosis is chronic strain from overexertion, and the treatment is rest. The child should not engage in athletics, particularly contact sports, until all signs and symptoms subside. A daily afternoon rest period should be prescribed. Not infrequently the child must be observed and treated for 18 to 24 months.

One injury to the lower extremity of the young child merits special mention, since the presenting symptom is usually limp. This is an undisplaced *fracture of the shaft of the fibula* in the middle or lower third. It is a fracture due to direct trauma. Sometimes an older sibling will ride his tricycle into the leg of the younger brother or sister. The child complains immediately of pain in the leg and is unwilling to bear weight upon the extremity. Careful physical examination will reveal tenderness to palpation over the site of injury, but this is often overlooked. Initial roentgenograms show no evidence of fracture. The child limps for a week or two, and the symptoms gradually subside. If films are repeated at 10 to 14 days after injury, the line of infraction through the fibula can be readily seen. Treatment is purely symptomatic. If the diagnosis is suspected early, the limb should be protected by a short-leg plaster boot.

THE LIMP OF HIP DISEASE

The hip is a deeply situated, stable, weight-bearing joint which is protected by strong ligaments and muscles. It is a ball-and-socket articulation with universal motion, but, because of its deep-seated anatomic location, the range of motion is less than in the shoulder. Many diseases involving the hip joint first manifest themselves by limp.

Toxic Synovitis

Among the commonest affections of the hip is an acute transient inflammation of the synovia which is known as a toxic synovitis. This is discussed more fully elsewhere in this symposium (p. 1073). The child will walk with an antalgic type of limp, flinching as the affected limb is put down and hopping quickly onto the normal leg.

Coxa Plana

Occasionally acute synovitis will be the first symptom of coxa plana, or Legg-Calvé-Perthes disease. This disease usually occurs in male chil-

dren between the ages of five and 12. The cause remains obscure, but the pathology is characterized by a disturbance in the circulation in the capital femoral epiphysis which results in sequestration of the epiphysis itself. The epiphysis passes through a phase of disintegration and of gradual repair. The progress of the disease from its inception to healing is slow and may take as long as four or five years. The most constant early sign is limp. The limp may be associated with muscular spasm and may or may not be painful. All motions of the hip joint are restricted. X-ray changes are usually characteristic.

Treatment consists in protected weight bearing either in bed in traction or in a nonweight-bearing splint until reconstruction of the femoral head is complete.

Congenital Dislocation of the Hip

Congenital dislocation of the hip is not a rare deformity. It is approximately six times more common in female infants than in males. Unilateral dislocation is more usual than bilateral. Unilateral dislocation in the young infant can be suspected by tightness of the adductor muscles and by slight shortening of the affected extremity. In addition, there is often absence of the normal postnatal flexion contracture of the hip.

After the child has begun to walk the instability of the hip joint results in a painless limp. The head of the femur is usually displaced posterior and superior to the acetabulum, causing instability of the hip joint and shortening of the extremity. Because the head of the femur is not situated in the acetabulum, the normal abduction power of the hip is disturbed, owing to absence of a fulcrum for the muscles to act upon. The gluteus medius muscle, the strong abductor of the hip, originates from the lateral aspect of the ilium and inserts into the greater trochanter of the femur. The mechanical efficiency of this muscle, so important in gait, depends upon normal location of the head of the femur in the acetabulum. In a dislocation of the hip the mechanical efficiency of the gluteus medius is disturbed. As the child places his weight upon the affected extremity, there is a tendency for the pelvis to drop. However, to maintain balance the child tends to throw his weight toward his involved side. In addition to the instability of the hip, the shortness of the leg contributes to the limp.

A thorough understanding of the Trendelenburg sign is of help in evaluating the limp in congenital dislocation of the hip. If the child is asked to stand upon the normal limb and raise the other leg, the opposite pelvis is raised to maintain balance over the weight-bearing line. However, if the hip is dislocated and the child is asked to stand upon the leg, it is impossible for the abductor muscles to raise the

pelvis on the opposite side, and hence it tends to drop. This is known as a positive Trendelenburg sign.

In bilateral dislocation of the hip the gait is typical and has often been described as a waddling limp. Because the weight-bearing line has been shifted posterior to the acetabulum, the child increases his lumbar lordosis to maintain the erect posture. He progresses forward, lurching from side to side with a prominent abdomen and a sway-back.

Treatment for congenital dislocation of the hip requires reduction of the head of the femur into the acetabulum. The reduction must be maintained by either a suitable splint or plaster cast, depending upon the age of the child and the stability of the reduction.

Coxa Vara

Waddling gait is also seen in coxa vara, a malformation of the upper femur characterized by a decrease in the angle of the shaft and the neck resulting in actual shortening of the extremity. In congenital coxa vara an osseous defect exists in the femoral neck which is usually bilateral. Aquired forms of the disease are due to rickets, osteomalacia, or as a complication of a growth disturbance secondary to fracture of the neck of the femur. The gait is similar to that seen in bilateral congenital dislocation of the hip.

LIMP DUE TO TUBERCULOSIS OF THE HIP JOINT

At the midpoint of the twentieth century tuberculosis is no longer referred to as the white plague. However, the disease has not vanished, and children are still afflicted with tuberculosis of the spine, hip and knee. One of the earliest and most classical signs of tuberculosis of the hip in the child is stiffness of the hip joint in the morning accompanied by limp. The limp will often pass off during the day, only to return toward evening, particularly if the child is fatigued. The lameness varies at first from day to day, and there are intervals when the child apparently walks normally. If the disease progresses unchecked, walking becomes difficult or impossible within a period of weeks or months.

In the earliest phase of the disease the hip joint is abducted and externally rotated as in this position the capsule is relaxed and symptoms are minimized. Because of this position an apparent lengthening of the limb occurs caused by abduction of the leg on the pelvis. The child is noticed to walk on his toe, placing the foot on the ground with great care. The heel is not brought down, since jarring causes intense pain in the hip. With progression of the disease the hip becomes adducted, internally rotated, and flexed on the pelvis. With this deformity the affected side drops as the child walks. Owing to pain on weight

bearing in the diseased hip, the child flinches and quickly puts his weight on the other leg.

Jones and Lovett,[3] in their classic textbook, have carefully analyzed each element of limp in tuberculosis of the hip. They point out that flexion of the hip is characterized by limp in which the body bends forward when weight is put upon the affected limb. If there is an adduction deformity, the leg is practically shortened, and lameness is most evident from the front in a lurch of the pelvis at each step. If the limb is abducted at the pelvis, there is practical lengthening, and the leg is carried out from the midline of the body in walking. Some lurching of the pelvis occurs at each step when the child is observed from the front. If the limb is shortened, the patient drops the pelvis on the affected side and leans the body to that side when stepping on the shortened limb. In tuberculosis the limp as observed is usually a combination of these various deformities.

Treatment of tuberculosis of the hip joint necessitates rest, not only of the hip specifically, but also of the body as a whole. This requires bed rest with traction or application of a plaster cast. In addition to measures directed toward improvement of health, chemotherapy with streptomycin, para-amino salicylic acid and the newer hydrazides of isonicotinic acid is administered. With improvement in the general health, surgical fusion of the hip joint should be carried out. Motion of the infected joint is incompatible with healing in tuberculosis, and the hip must be placed at permanent rest by surgical fusion.

THE LIMP IN SLIPPING OF THE CAPITAL FEMORAL EPIPHYSIS

This disease, also known as epiphysial separation or fracture, epiphysial coxa vara, and femoral osteochondritis of adolescence, is a potentially crippling disorder. Special emphasis is placed upon early diagnosis. Howorth[2] has described four stages in the disease: preslipping, slipping, healing, and residual. Limp is a cardinal symptom of the preslipping phase of the disease, and it is during this phase that the diagnosis should be made. Slipping of the capital femoral epiphysis is an affection of the hip which comes on between the ages of eight and 19, with an average age of about 12 or 13 years. In Howorth's series the average age of slipping is approximately 13.5 years. The average age of slipping for girls was two years less than for boys. The majority of the children are either large for their age or are passing through periods of extemely rapid growth. They do not have endocrine, dietary or vitamin deficiencies or imbalances. Trauma when associated with the history is usually of the most trivial sort. The exact cause of the disease remains obscure.

In the preslipping stage the pathology consists of a synovitis of the

hip with hypervascularity at the junction of the neck of the femur and the epiphysial plate of the head. In the slipping state the epiphysis is displaced downward and backward in relation to the femoral neck.

Often the only sign of the preslipped epiphysis is limp. The limp may be painless, and the child is simply noted to drop his shoulder toward the affected side as weight is placed on the limb. The symptoms, somewhat like those in early tuberculosis of the hip, are intermittent at first. The limp may be present for a few days and then may disappear. The symptoms of preslip may last several weeks or several months. As the disease progresses, the limp becomes more protective and is due to pain and muscle spasm. Physical activity aggravates it, and it is particularly severe after engagement in athletics. As the epiphysis migrates posteriorly in the progression of the disease, some element of shortening of the extremity enters into the limp. In addition to pain, the child is aware of shortening in the extremity. Pain is often referred along the course of the obturator nerve to the inner side of the knee or thigh. Many an unsuspecting physician has x-rayed knees repeatedly when the abnormality was to be found in the hip.

Once the diagnosis is made, the child should be removed promptly from all further weight bearing upon the affected limb. *Therapy* is dictated by the degree of slip, and there is considerable variation of opinion as to precisely what measures should be carried out. It suffices to say that treatment should be directed toward prevention of further slip and toward prompt closure of the epiphysial plate between the head and neck of the femur. This can be accomplished by placing small bone grafts across the epiphysial line or by using various types of metallic intramedullary fixation. The child should be kept on crutches until the epiphysial line is closed. This will usually take place within several months.

PARALYTIC LIMP

Poliomyelitis

Chronic poliomyelitis with involvement of the musculature of the trunk and lower extremities is the most common cause of paralytic limp in the child. From the careful examination of the gait the physician trained in musculoskeletal disorders is able to determine which muscle or muscle groups are affected and can also judge what measures can be taken to improve locomotion. Careful study of the patient's gait is the first and possibly the most important step in the examination. Certain muscles in the lower extremities are affected more frequently by the virus of poliomyelitis than others, and it is the abnormalities associated with weakness in these that will be discussed in detail.

The gluteus medius, the strong abductor of the limb at the hip, is

frequently paralyzed by poliomyelitis. The gait associated with weakness or paralysis of the gluteus medius is perfectly characteristic. When the patient bears weight on the weakened side, he lurches over toward that side in an effort to maintain balance. The limp is similar to that due to shortening of the extremity or to congenital dislocation of the hip or coxa vara.

The gluteus maximus is the strong extensor muscle of the hip joint. It is one of the most important muscles in the support of the trunk upon the lower extremities. In paralysis of the gluteus maximus the body is thrown backward with a lurch when weight is borne upon the affected limb, and the patient hastens to place the other foot forward. The limp is similar to that seen in persons who wear an artificial limb for a high thigh amputation.

In paralysis of the flexor muscles of the hip joint the normal forward progression of gait is disturbed. The patient has great difficulty raising the limb at the hip and hence progresses forward by an excessive rotation of the pelvis on the affected side.

Complete paralysis of the quadriceps femoris muscle makes walking extremely difficult. The quadriceps femoris is the strong extensor muscle of the leg on the thigh. The patient has to compensate for loss of the extensor of the knee by actively hyperextending the knee by placing the hand on the front of the thigh to prevent a fall from sudden flexion of the knee joint. The patient also may tend to compensate by walking with the limb in external rotation. If the hamstring muscles are strong, he is able to lock the knee without hyperextension or even by fully extending it by simply bending the whole body forward and thus carrying the center of gravity forward over the leg.

In paralysis of the gastrocnemius the gait loses spring. The walk becomes heavy and awkward, and the heel is brought down hard. If one side only is involved, the normal rhythm is disturbed and the patient tends to pause for a longer time on the well leg. If both sides are weak, the feet are pulled upward at the ankles and the patient has a waddling type of gait.

In paralysis of the anterior musculature of the leg, particularly of the tibialis anticus, the patient has a foot drop and, in order to clear the toes, has to lift the limb from the ground much higher than is usually necessary. If there is a contracture of the heel cord associated with weakness of the anterior musculature, the patient is required to walk on the toes, and forward progression is extremely difficult. With a bilateral heel cord contracture, walking is almost impossible.

The gait of the child afflicted with chronic poliomyelitis is often a component of the elements due to weakness in one or several of the muscles described. It is important to analyze gait carefully to determine

precisely how much disability the child has and also to determine what can be done to make walking simpler and less arduous.

Treatment in chronic poliomyelitis depends, of course, upon the extent of weakness or paralysis. Compensation for loss of, or replacement for, the supporting musculature of the hip is extremely difficult.

Loss of the gluteus medius power is one of the most difficult to treat. Muscle transplants are on the whole ineffective, and crutches offer the only adequate means of support. For weakness below the hip suitable bracing is helpful in facilitating walking. Transfer of tendons, stabilization of affected joints by various procedures, release of contractures, and epiphysial arrests all have a place in the treatment of the chronic paralytic.

Cerebral Palsy

Limp is often a sign of mild cerebral palsy. Of course the limp depends upon the extent of involvement and upon whether the affected muscles are normal, spastic or weak. Also, the type of limp depends upon the nature of the palsy, whether it be athetoid, spastic, ataxic or mixed. A child with a mild spastic type of cerebral palsy may have strong posterior but weak or normal anterior calf muscles, tight hamstrings and flexion deformities of the knees and hips. These children tend to walk with their knees in slight flexion and, owing to the tightness of the heel cords, are unable to bring the heel to the ground in forward progression. Those with spastic adductor muscles and weak abductor muscles have a typical scissors type of gait in which the knees touch or cross as the child walks. The hips are flexed, internally rotated and adducted. Both knees are held in flexion and the feet in equinus.

Intelligent *treatment* necessitates careful evaluation, including a muscle examination. Lengthening or section of contracted tendons, braces, muscle re-education and gait training are all part of the treatment of the child with cerebral palsy.

Spina Bifida

The limp in spina bifida is due to muscle weakness. In spina bifida associated with myelomeningocele the extent of weakness is greater than in the occult types. Frequently adhesions in the region of the cauda equina make traction upon the cord. As the spinal column grows, the cord passes through a normal upward progression. Firm adhesions make traction upon the cord and hinder this upward progression. The fixation of the cord is manifested clinically by a changing muscle picture.

Treatment should always be conservative, and surgical procedures should be left until growth is complete. The limp depends entirely upon which muscles are involved.

Charcot-Marie-Tooth Disease

In Charcot-Marie-Tooth disease, or hereditary muscular atrophy of the peroneal type, limp is an early sign. The disease is due to degenerative changes in the peripheral nerves of unknown cause. It makes its appearance usually between the ages of five and 10, and boys are affected more frequently than girls. There is a familial tendency to the disease. The earliest signs are slight shortening of the heel cord and inversion, or varus, of the foot. The child walks on the outside of the foot with an unsteady stumbling gait in which he is unable to bring his heel to the ground. The lateral stability of the foot is disturbed, owing to weakness of the peroneal muscles, and frequent falls are characteristic.

Treatment consists in correction of the contracture of the heel cord and in restoration of lateral stability of the foot by suitable bracing or by arthrodesis of the foot.

Friedreich's Ataxia

Friedreich's ataxia is a progressive familial disease which usually develops early in childhood. Approximately 90 per cent of cases appear before the fifteenth year. Children develop weakness of muscles in the lower extremities and walk with their feet placed widely apart with a swaying, irregular type of gait.

Progressive Muscular Dystrophy

Progressive muscular dystrophy also begins in early or middle childhood and affects males more frequently than females. An apparent hypertrophy of the muscles of the calf occurs due to deposition of fat between the muscle fibers. The earliest sign is often contracture of the heel cord, which causes the child to walk on his toes. Weakness in the legs, easy fatigability, and limp are early symptoms. As the disease progresses, the gait becomes peculiar. The patient leans back from the hips, sways a bit, and balances carefully in progressing forward. In the early and middle stages of the disease the dominating factor is weakness of the extensor muscles of the lower extremities. Because of weakness of these muscles, the child has great difficulty arising from the floor.

Treatment should be conservative. These children do poorly if placed in bed for long periods of time, and rapid, progressive weakness de-

velops on complete rest. Surgical lengthening of the heel cord is of value at times. Some benefit has been reported from the use of folic acid and protein hydrolysate supplements in the diet.

SUMMARY

Locomotion is a most important function of the lower extremities. Impairment of locomotion, whether it be due to bony abnormality, muscular weakness or primary neuropathy, always manifests itself by a variation in gait. The importance of limp as a cardinal sign of disease of the skeleton and its associated structures has been stressed. Careful and repeated observation of the lame child is of importance not only in arriving at an early correct diagnosis, but also in the evaluation of the progress of disease.

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LESIONS OF THE KNEE IN CHILDHOOD

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In childhood the knee is the site of more pathologic disorders than any other articulation in the body. Infectious processes, malignancies and metabolic disorders may be encountered; these do not differ from those observed in other joints. Its exposed position renders the joint vulnerable to severe single trauma and to repeated minor trauma incident to function. Static abnormalities such as flat and pronated feet and faulty postural habits may be responsible for the development of deformities of the joint such as genu valgum and genu varum. Finally, numerous osteochondropathies are frequently observed in this joint. In this presentation some of the more common deformities and osteochondropathies of the knee are discussed.

OSTEOCHONDRITIS DISSECANS

The designation "osteochondritis dissecans" is applied to loose bodies in joints arising from the articular surfaces of the ends of the bones. Detachment does not occur suddenly, but may require months or even years before it is achieved. In some instances detachment does not occur, and spontaneous healing may result. This disorder must be distinguished from that in which loose bodies are produced by violent injury which are really comparable to subchondral fractures with immediate displacement of the fragment into the joint cavity.

The designation "osteochondritis" was first used by Konig in 1887. The knee joint is most frequently implicated, and in my experience 92 per cent of all cases of loose bodies from all known causes were due to osteochronditis dissecans. Generally one joint is implicated; occasionally both knee joints are involved.

Etiology and Pathogenesis

Although many observers believe that trauma plays the most significant role as a causative agent, sufficient clinical evidence has not been advanced to support this premise. The most plausible explanation of the disorder was proposed by Phemister. He notes that the articular cartilage is devoid of blood supply and that there is little sensation in subchondral bone. Hence it is possible for trauma to produce an incomplete subchondral fracture without pain or other clinical manifestations of sufficient severity to focus the attention of the patient on the incident.

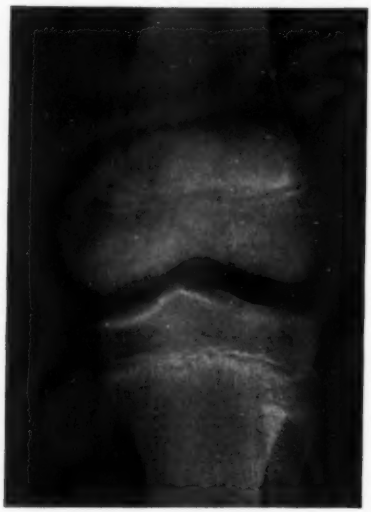


Fig. 70. Osteochondritis dissecans implicating the medial femoral condyle of a boy 9 years of age. Observe that the affected area in the condyle is clearly visible and that a necrotic portion of the condyle lies in a well demarcated niche.

In the majority of instances the lateral aspect of the medial femoral condyle, close to the insertion of the posterior cruciate ligament, is involved. In a few cases the lateral femoral condyle is implicated, and occasionally the lesion is found in both condyles. In the early stages of the disorder the lesion implicates a small circumscribed area of the femoral condyle and is comparable to an incomplete subchondral fracture. At this time there is no break in the continuity of the articular cartilage; the only gross evidence of the lesion is some softening and discoloration of the involved area; in some instances even these features are absent, and the lesion can be detected only by careful roentgenologic study (Fig. 70). In untreated patients union does not occur because the circulation to the affected fragments is severely impaired.

In many of these cases repeated trauma incident to normal function is sufficient to preclude normal healing between the parent bone and the fragments. As the result, degenerative alterations both in the cartilaginous and osseous elements of the fragments increase in severity, and eventually extrusion of the fragments into the joint cavity occurs. Before extrusion occurs the cartilaginous surface is soft and frayed and exhibits a yellowish color. At this time the loose bodies can be readily shelled out of their niche in the femoral condyle. Upon enucleation of

the fragments one notes that the opposing surfaces are covered with fibrous tissue and fibrocartilage. As long as the body remains in the niche it still retains some attachment by a fibrous pedicle. Upon complete severance of all attachments the degenerative alterations in the loose body increase in intensity. The osseous elements undergo necrosis, and the articular cartilage reveals marked fibrillation, disintegration and calcification. The free body may eventually become invested completely in fibrocartilage. In some instances the body may increase in size because it may obtain sufficient nutrition from the synovial fluid. Eventually the loose body may come to rest in one of several places in the joint: namely, in the suprapatellar pouch, in the intercondylar notch in front of the anterior cruciate ligament, or it may gravitate into the posterior aspect of the joint. Not infrequently the body may move about in the joint and give rise to symptoms consistent with internal derangement of the knee joint.

Clinical Features

This disorder is frequently observed in late adolescence. Ninety per cent of the cases affect males. As a rule, one knee is implicated, but in a small percentage of cases the lesion is bilateral. The general health of the patient is usually excellent. In some instances a specific history of trauma is elicited, but in most instances no such history can be obtained. The clinical features are governed by the position and size of the loose body. In most instances children are aware of some vague pain in the joint which is accentuated by activity and relieved by rest. Not infrequently the episodes are accompanied by varying degrees of swelling. These symptoms are usually noted in patients in whom the affected area of the articular cartilage still remains in its niche in the parent bone. The episodes noted above increase in intensity until the fragment is extruded into the joint. The clinical manifestations are governed by the behavior of the loose body.

Loose bodies floating around in the joint may give rise to symptoms of "giving away" and momentary locking of the knee joint followed by effusion. Not infrequently the patient will volunteer the information that he is aware of something moving about within the joint, and often the body assumes a position between the articular margin of the joint and the capsule; now a hard, bony mass may be palpable at the joint margin. Synovial effusion is a common feature of this disorder. Occasionally the body may develop a pedicle at its new site of attachment, allowing sufficient mobility so that the body may be trapped momentarily between the articular surfaces. Repeated episodes of "giving away" and effusion may cause varying degrees of laxity of the quadriceps

mechanism, and synovitis, and, in patients with numerous episodes, the knee becomes lax and exhibits persistent synovial and periarticular thickening.

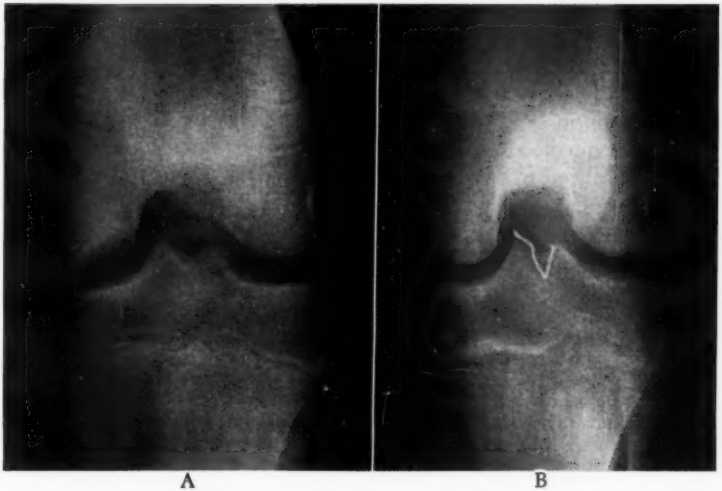
Physical examination reveals varying degrees of atrophy of the quadriceps muscle. As previously noted, the episodes of locking and instability are usually associated with an effusion. Therefore swelling is readily demonstrable. On several occasions I have been able to palpate the free body at the articular margin of the joint. Roentgenographic studies invariably establish the diagnosis. Roentgenograms not only locate the position of the body in the joint, but also depict the crater or niche in the femoral condyle from which the loose body originates. In very early cases, however, one must remember that roentgenograms may fail to show evidence of any abnormality in the articular cartilage, or, if the detached fragment is composed entirely of cartilaginous tissue without calcification, will fail to provide sufficient information about the true nature of the lesion. Roentgenograms fail to establish the actual size of the free bodies because much of the body may comprise cartilaginous tissue without calcification. On the other hand, roentgenograms may show a wide zone of absorption in the cancellous bone between the parent bone and the affected overlying area.

Treatment

Conservative Management. The management of this entity must be governed by the stage of the pathologic process present at the time treatment is instituted. It has now been proved that children in whom the fragment has not yet separated respond favorably to conservative treatment. Many workers are prone to excise the area affected regardless of whether or not the affected portion of the condyle is separated from the host bone. However, sufficient clinical evidence has been offered to indicate that some cases of osteochondritis dissecans heal spontaneously. Six such instances were reported by Weiberg in 1943; he concluded that conservative measures were indicated and should be instituted in patients up to the age of 16 or 17 and, in some cases, up to the age of 20, and if, during the period of treatment, the fragment became detached, it should be removed. This has been my experience. I have had occasion to see 11 such cases in which surgical intervention was not done and the patients were treated by conservative therapy. The age of these patients ranges from 12 to 16 years. This has also been the experience of other workers who have reported similar cases in the literature. In the light of this information it becomes apparent that in growing children in whom reparative processes are at a high level, osteochondritis dissecans without separation of the fragments should be given a trial at conservative treatment (Fig. 71).

On the other hand, in cases in which the crater in the articular surface is sclerotic and lined by mature fibrous tissue, and in instances in which the fragment is attached only by a pedicle, conservative measures are doomed to failure. Here surgical intervention, whereby the body is excised, is the treatment of choice.

Conservative intervention comprises the application of a nonpadded plaster cylinder extending from above the malleoli to the groin with the knee extended. The patient is ambulatory on crutches. The heel of the



A B

Fig. 71. A, Osteochondritis dissecans of the medial femoral condyle of a boy 10 years of age. *B*, This knee was treated by immobilization for a period of 5 months. Note that complete healing has taken place without extrusion of a loose body.

shoe on the opposite foot is elevated so that the foot on the affected side does not touch the floor. Throughout the period of treatment the quadriceps muscle is exercised on a regulated regimen. In most instances a new plaster cylinder is applied every four to six weeks. At the end of three months, roentgenograms are taken to note the progress of the healing. If vascularization is demonstrable, but healing is not complete, immobilization is continued for two or three more months. On the other hand, if the body becomes detached during this period, surgical intervention is indicated.

Surgical Management. It is important that roentgenograms be taken immediately before the operation is performed. In order to preclude changes of the position of the loose body, the position of the limb must not be altered after the final roentgenograms have been taken. It is essential to recall that the calcified bodies and those containing some

osseous tissue are readily depicted by roentgenograms. However, this method of study may fail to reveal the presence of loose cartilaginous bodies. Hence a thorough search must be made of the regions of the joints explored at the time of operation, particularly those regions which are most likely to harbor the bodies, such as the intercondylar region and the suprapatellar pouch.

In view of the difficulties that may arise in locating the loose bodies, small incisions are not justifiable. The incision should be adequate and provide the surgeon considerable freedom in locating the fragments. In most instances, portions of the articular cartilage at the periphery of the crater are fragmented and loose. Failure to remove this tissue may result in the formation of more loose bodies and recurrence of symptoms. On the other hand, unnecessary extensive exposures should not be made routinely. Long parapatellar incisions are not necessary to expose the mediolateral compartments of the knee joint when the lesion is localized to one of the femoral condyles. Parapatellar incisions should be made only when concomitant pathology of the synovial membrane is present which may require partial synovectomy in addition to the removal of the loose fragment. Finally, before the wound is closed, roentgenograms should be taken with a portable unit to detect whether or not all radiopaque bodies have been removed.

The management of the affected area varies in different cases. In the event that the body lies loosely in the defect, it should be removed. This is best achieved by a sharp-curved gouge which readily scoops the fragments out of the niche. Soft and fibrillated articular cartilage bordering the defect is shaved off carefully in layers until normal cartilage is reached. Finally, the edge of the crater is bevelled. Some attention should be paid to the floor of the crater. With a sharp curet all debris is removed from the floor of the defect until normal bleeding and cancellous bone are exposed. Old cases may exhibit sclerotic bone lining the floor of the crater. In such instances all loose connective tissue is removed with a sharp curet and the sclerotic bone is perforated by numerous fine drill holes. This step assures revascularization of the sclerotic bone and allows the crater to fill with blood, thereby initiating the formation of fibrocartilage in sufficient quantities to obliterate the cavity. Occasionally the loose bodies are found at a distance from the niche in the articular surface, lying either free or attached to the synovial lining. The body is first removed and then the site of origin is located and treated in accordance with the circumstances present.

Although it is not the purpose of this presentation to describe technical surgical procedures, it is pertinent to mention that loose bodies arising from the medial condyle and lying in the anteromedial compartment and in the intercondylar notch are made readily accessible through an anteromedial incision. If the fragment is in the posteromedial compartment, extension of the posterior limb of this incision to a point 1 cm. posterior to the medial epicondyle of the femur is sufficient to give access to the posterior compartment, which is reached through a second capsular incision made behind and parallel to the longitudinal fibers of the superficial portion of the tibial collateral ligament.

Loose bodies arising from the laterofemoral condyle are removed in a similar manner as those arising from the medial femoral condyle. In these instances an anterolateral curve incision is made to expose the anterolateral compartment, and extension of this incision posteriorly permits access to the posterolateral compartment.

In all instances the suprapatellar pouch should be inspected for the presence of bodies not depicted in the roentgenogram. Inspection of this pouch is readily made

by the two afore-mentioned incisions. Digital examination of the pouch is permissible in making a thorough examination of the suprapatellar pouch.

Occasionally it may be desirable to limit the exposure to the posteromedial or posterolateral aspect of the joint. In such cases the posteromedial or posterolateral incisions of Henderson provide the desired exposure. These incisions are especially useful for removal of loose bodies from the posterior compartment.

The postoperative management comprises the application of an elastic compression bandage immediately after operation is completed. The bandage extends from midcalf to midthigh, and the limb is put at complete rest in order to preclude filling of the joint with blood from the raw bony surfaces. In spite of compression, however, bleeding may occur. In such instances aspiration should be done as frequently as necessary in order to prevent pain and distention of the soft tissues of the joint.

Immobilization is continued for two or three weeks, depending upon the intensity of the joint reaction. At the end of this period quadriceps drill and flexion exercises are instituted. Weight bearing should not be permitted for at least three weeks postoperatively. Quadriceps exercises should be continued until the maximum level of efficiency, comparable to that of the normal quadriceps, has been reached. Removal of bodies from the posterior compartment through posteromedial, posterolateral or posterior midline incisions does not require a long period of delay before rehabilitation is commenced. Active motion at the knee joint and quadriceps exercises are started immediately after the operation.

Occasionally bodies arise from other regions of the knee joint than the medial femoral condyle. They may arise from the lateral condyle of the femur, the patella and, in many instances, from the tibia. Therefore it becomes apparent that, in the event that no defect is found in the medial femoral condyle, other areas of the joint should be investigated. In most instances, however, roentgenographic studies usually depict the site of the lesion.

OSGOOD-SCHLATTER'S DISEASE

Etiology and Pathogenesis

Much controversy still exists as to the etiologic factors responsible for this entity. Many observers are of the opinion that repeated stresses applied to the tibial tubercle produce minor avulsions of fragments of the tubercle or of the entire ossification center. However, some of the clinical features of this entity fail to support this view, particularly the frequent bilateral occurrence. Also, in the same age period, similar changes are often noted at the site of insertion of the tendo achillis to the os calcis or at the site of insertion of the peroneus brevis into the base of the fifth metatarsal bone and, finally, in the upper and lower poles of the patella.

The most plausible theory has been set forth by Ferguson,[4] who is of the opinion that the bony changes noted in Osgood-Schlatter's disease are secondary to swelling and hemorrhage and degenerative changes in the patellar tendon near its attachment. These changes interfere with circulation of the underlying bone and cause irregular ossification within it. He designated this process as tenonosis, which is caused by stress developed by rapid growth and expended on the patellar tendon near its attachment during an active period of growth. The same theory can be applied to the changes noted at the poles of the patella described by Larson and Johanson. According to Ferguson, irregular ossification in the tibial tubercle appears some months after swelling of the tendon has subsided and tends to undergo repair after a few years. Frequently these sites of ossification are interpreted as fractures or avulsions of the tubercle.

Clinical Features

This entity is observed in adolescence. Boys are more frequently affected than girls, and frequently it is bilateral. It is characterized by

Fig. 72. A typical case of Osgood-Schlatter's disease implicating the tibial tubercles of both legs. Note the marked prominence on the anterior aspect of the tibia and swelling of the patellar tendons.

varying degrees of swelling of the tibial tubercle and some swelling and thickening of the distal end of the parapatellar tendon (Fig. 72). Pressure over the tubercle and tendon near its insertion invariably elicits considerable tenderness. The symptoms usually come on insidiously, and the patient, in many instances, is only aware of dull pain over the anterior surface of the knee joint which is accentuated by activity and relieved by rest. At times a limp may be discernible, and, in the more

acute cases, pain is felt when the leg is extended against resistance. Roentgenographic studies disclose in the early cases only thickening of the parapatellar tendon, anterior to the tibial tubercle, and some swelling of the overlying skin and subcutaneous tissue. Early in the process irregular ossification is observed in the tibial tubercle, and some calcification may be noted in the substance of the patellar tendon (Fig. 73). However, we must remember that not infrequently, irregular

Fig. 73. Roentgenogram exhibiting typical features of Osgood-Schlatter's disease in both the right and left lower legs.

ossification may exist without symptoms. In such instances, however, the patellar tendon shows no evidence of swelling, and the irregular ossification may be the result of multiple centers and not of the process we are describing.

This entity is a self-limited process. It may persist for a few months or for two or three years. However, invariably, all symptoms subside, but in most instances the affected tubercle remains slightly larger and more irregular than normal.

Treatment

The clinical signs and symptoms observed in most instances do not justify enthusiastic treatment of this lesion. I am of the opinion that if the parents and the patient are made aware of the course and the benignity of the lesion, in most instances only palliative measures are required. In my experience the parents are mostly concerned with the presence of a lump on the anterior aspect of the knee joint whose

identity has not been thoroughly established in their minds. Furthermore, I do not believe that these cases should be treated routinely by means of immobilization or surgical intervention.

Most cases can be treated by limiting the patient's activity when accentuation of symptoms occurs and applying a protective dressing over the tubercle. This is best achieved by applying a piece of felt or sponge rubber with a hole in the center over the tubercle and holding it in place by means of an elastic bandage. The bandage should not be worn continuously. However, I have encountered a few cases in which the symptoms were of unusual severity. All motions of the knee were acutely painful, and the tubercle exhibited marked swelling and acute tenderness. In such instances rest in bed until the acuteness of the symptoms has subsided is justifiable. Also, immobilization in a plaster cylinder affords the desired relief. Generally, a period of 10 to 12 weeks of immobilization in a plaster cast effects a cure. However, *this method of immobilization applies to only a few selected cases.*

Surgical management is limited to a few cases in which the severity and the duration of the symptoms demand that more radical measures be instituted. Many surgical procedures have been used to effect a cure. Among these are stapling of the tendon, multiple drilling of the tibial tubercle, pegging with a bone graft and excision of the loose fragments beneath or in the patellar tendon. Ferciot and Thomson[3] advocate complete excision of the tibial tubercle in order to avoid the deformity frequently noted in the severe cases.

In my hands splitting of the patellar tendon has been a successful and gratifying procedure. The tendon and tubercle are readily exposed by a J-shaped incision placed to one side of the midline and extended from the level of the apex of the patella to the distal end of the tubercle. Four to six parallel longitudinal cuts are made in the tendon penetrating approximately half of its thickness. At the termination of the operation a plaster cylinder is applied. Inasmuch as the continuity of the extensor mechanism is not interrupted, ambulation, with weight bearing, may be instituted seven to 10 days after the operation. At the end of three weeks the cast is removed and exercises are instituted to restore normal joint function.

GENU VALGUM (KNOCK KNEE)

This deformity of the knee is frequently encountered in childhood. It comprises an inward projection of the knee joint and a lateral deviation of the leg in relation to the longitudinal axis of the femur, forming an outward angle of varying degrees. Other secondary changes may be noted in long-standing cases. These comprise rotation outward of the tibia on the femur, resulting from prolonged traction of the biceps

femoris and the tensor fasciae femoris muscles. Occasionally the tibia may develop a compensatory varus deformity, and finally the distal end of the femur may exhibit an outward twist.

Etiology

The deformity may be the result of numerous causative agents. At one time rickets was believed to be the most frequent cause. However, it is now appreciated that rickets is not a primary cause and that, in most instances, congenital and hereditary factors may be responsible for the disorder. The most frequent causes are static abnormalities. Notable among these are pronated and flat feet and obesity. Finally, trauma to the epiphysial cartilage plate may produce a disturbance in growth which results in valgus deformity. In this presentation only those forms of genu valgum will be considered which are not associated with rickets or other malacic diseases or to trauma.

Clinical Features

Under the age of five many children exhibit mild degrees of genu valgum. As a rule, the disorder is physiologic and not associated with bony, endocrine or dietary disorders. A normal child in this age group stands with the medial femoral condyles and the internal malleoli in close proximity, almost touching. This is not true of children with genu valgum. These patients disclose, on standing, that the medial femoral condyles touch one another and that the internal malleoli are separated. The knees rub together in walking, and the line of weight bearing no longer passes through the center of the knee joint, but is shifted to the outer side, resulting in a rather awkward, lurching gait. If these deformities continue without correction, the supportive tissues, particularly on the inner side of the knee joint, are subjected to abnormal stresses which produce laxity of the structures and, not infrequently, a chronic synovitis of the knee joint. It is interesting to note that in this group of patients, when the knee is flexed, the deformity disappears. This is explained by the fact that in flexion the tibia articulates with the posterior portions of the femoral condyles which are not affected.

Treatment

In growing children conservative measures should be taken in all mild cases. Much can be done by attempting to restore the normal line of weight bearing, and to correct the pronation of the feet which, in most instances, is a concomitant finding. This can be achieved by a

medial heel wedge and a sole wedge from ⅛ to ¼ inch, depending upon the child's age. Manipulative measures have much to offer. The parents should be instructed properly in the execution of the manipulative maneuvers.

If the right leg is affected and is to be manipulated, the parent grasps the distal end of the femur immediately above the condyle with the left hand, and the right hand grasps the leg just above the malleolus. The leg then is forced repeatedly into adduction. By so doing, the soft tissues on the lateral aspect of the knee are stretched, and, at the same time, intermittent pressure is applied to the medial femoral condyle. In some instances it may be wise to apply night splints which tend to maintain the lower leg in normal alignment with the femur. In more resistant cases, braces should be prescribed.

Only rarely is surgical intervention indicated in cases which are not the result of trauma to the epiphysial plate. If, after a conservative trial, the deformity is such that it is not accepted clinically, then, of course, some form of surgical intervention should be considered. The most efficient procedures to correct angular deformity, such as noted in genu valgum, are stapling of the epiphysial plates of the distal end of the femur and upper end of the tibia on the medial aspect, or osteotomy at the site of maximum deformity.

GENU VARUM (BOWLEGS)

Bowleg deformity is a frequent finding in children under the age of four. The deformity is characterized by a lateral displacement of the knee joint so that the joint lies to the outer side of the line of weight bearing dropped from the anterosuperior spine of the ilium. As a rule, the lesion implicates the tibia or the femur, and, in most instances, the alterations in the bones of the lower leg are usually of greater severity than those observed in the femur. A frequent concomitant finding in the lower leg is an internal twist or torsion from the knee to the ankle (Fig. 74). This torsion implicates the tibia, forcing the toes to turn inward. If the child stands erect with the feet together, the deformity is readily recognized. The medial borders of both extremities form an O, and the knees are widely separated.

Etiology

Many cases of genu varum, which have been previously designated as of congenital origin, are the result of the cross-legged position that the child assumed in utero. Other etiologic factors are faulty postural habits which the child develops during growth. In many instances children sleep on the abdomen and face with the toes turned in and

the knees widely separated. As previously noted, mild or moderate bowing is frequently encountered in children under four years of age.

For completeness, other factors must be mentioned, namely, malacic bone diseases, particularly rickets, which predisposes the patient to this angular deformity when static factors are in force. In such instances the effect of body weight is sufficient to produce bending deformities, and, in some instances, stress factors produce fractures which may heal in malposition. Fortunately, rickets is not a frequent disorder of chil-

Fig. 74. Back and front views of a child 4 years of age exhibiting bilateral bowlegs. Also note the internal torsion of both tibias and a tendency for both forefeet to go into mild pronation.

dren in this country, and when it does occur, it is recognized early and treated adequately before deformities occur. Factors which cause arrested growth of the medial portion of the upper tibial epiphysial plate may produce this deformity, such as trauma or a form of osteochondritis implicating the inner aspect of the tibial epiphysial plate. Finally, bowlegs is a common deformity encountered in cases of faulty and chondral ossification, dyschondroplasia and in endocrine disturbances. This latter group interferes with the normal growth of the tibial and femoral epiphysial plates, particularly the medial portions, or there may be cessation of growth of these portions of the plates.

Clinical Features

As previously recorded, when the child stands with feet together, the knees are widely separated and the inner aspects of the extremities

form a symmetrical O. The child exhibits a waddling gait not unlike that encountered in bilateral dislocation of the hip. When the deformity is a concomitant finding of dyschondroplasia and rickets, the clinical features of these entities are manifested.

Treatment

It is generally recognized that mild deformities in children under the age of four, which are on a physiologic basis and not associated with bone, metabolic or endocrine disorders, in most instances will be spontaneously corrected with or without treatment. Many of us have observed idiopathic bowlegs in early childhood become knock knees after the age of four. However, measures should be instituted which may enhance correction of the deformity. This is applicable to patients under and over the age of four years. These measures comprise inner wedges on the heel and sole of the shoes and manipulative maneuvers which tend to straighten the bowing. Forceful manipulation should not be performed because it may inflict damage to the epiphysial plate. Manipulative maneuvers should be performed regularly, 10 or 15 times once or twice daily. If these measures fail, braces should be applied.

Only rarely do bowlegs of physiologic origin require surgical intervention. However, this is not true of severe residual deformity of rickets in childhood or of renal rickets, such as is manifested about the age of puberty, or in severe deformities from other causes. One must be sure that no surgery, in these cases, is performed during the active stage of malacic diseases. Corrections at this time are doomed to failure, and recurrence is the rule.

TIBIA VARA (OSTEOCHONDROSIS DEFORMANS TIBIAE)

This disorder is more common than is generally recognized and should be distinguished from other causes capable of producing angular deformities of the knee joint. The pathology implicates the metaphysis and epiphysial plate and also the osseous center of the epiphysis. The alterations are located on the medial side of the proximal tibial epiphysis and produce an abrupt varus angular deformity immediately below the epiphysis. Concomitant findings comprise varying degrees of genu recurvatum and internal rotation of the leg. Blount[1, 2] described two forms of tibia vara, the infantile and the adolescent.

Infantile Type

This form is encountered in children after the first or second year of life who, otherwise, are of normal development. However, in many

cases overweight is a concomitant finding. With increase in growth, the varus deformity becomes progressively worse. In 50 per cent of the cases the lesion is bilateral. As yet no responsible etiologic factor has been uncovered. There is some evidence that a congenital factor may be responsible in the infantile type.

The roentgenographic features are typical of the lesion (Fig. 75). They comprise a sharp angular deformity immediately below the

A B

Fig. 75. Roentgenograms of a case of infantile tibia vara noted in a child 2 years of age. *A*, Observe the angular deformity immediately below the proximal tibial epiphysis, wedging of the osseous portion of the epiphysis, and the prominent beak on the medial portion of the metaphysis. *B*, An oblique view of the same knee joint. Observe the prominence of the beak on the medial and posterior aspects of the metaphysis of the tibia.

proximal tibial epiphysis. The medial portion of the epiphysial plate may be expanded and irregular. The osseous portion of the epiphysis is wedge-shaped with the base placed laterally. The medial portion of the metaphysis is prominent, resembling a beak; irregular cartilage islands are observed in the beaklike projection of the metaphysis. Finally the prominent position of the metaphysis projecting medially is covered by hyaline cartilage.

Pathology

In all instances there is an abnormal growth of the epiphysial plate and retarded ossification of the medial portion of the tibial epiphysis. A beaklike projection is noted in the region of the inner aspect of the metaphysis and appears to function as a buttress under the epiphysis. Here, irregular areas of cartilage are depicted on the roentgenogram as

areas of decreased density. Microscopically, the pathology in this area does not differ from that observed in chondrodysplasia.

Fig. 76. Roentgenogram of a typical case of adolescent tibia vara in a patient 12 years of age. Note that the varus deformity is most prominent immediately below the epiphysial plate. It appears as if some arrest of the medial portion of the proximal tibial epiphysis has occurred.

Adolescent Type

This form of the disease is noted in children between the ages of six and 12 years who, otherwise, show no abnormal disturbance of growth. In the adolescent the lesion is usually unilateral, and many observers believe that trauma may play a part in initiating this form of the disease. The roentgenographic features suggest an arrest of growth of the medial portion of the proximal tibial epiphysis producing the varus deformity immediately below the epiphysial plate (Fig. 76). The epiphysial plate shows some irregularity and may be more narrow on the medial side than on the lateral side. Occasionally the epiphysial plate in this area may be obliterated. An abrupt angular deformity is found below the metaphysis. The apex of the deformity is placed laterally.

Clinical Features

The infantile type is usually bilateral in 50 per cent of the cases, whereas in the adolescent type the lesion is usually unilateral. The general condition of the patient is excellent, and the onset of the disease is an insidious one, with gradual increase in development of the varus deformity without clinical manifestations or local or systemic disorders. Spontaneous correction may occur in the infantile type. These patients often exhibit a waddling gait, whereas, in the adolescent type, the patient may walk with a definite limp. Because the foot and knee on the affected side are subjected to abnormal stresses incident to function, these patients may exhibit symptoms of strain of the knee joint.

Upon inspection one notes a sharp angular deformity immediately below the knee and, in most cases, varying degrees of genu varum,

internal rotation of the tibia on the femur and pronation of the foot. The medial side of the joint exhibits varying degrees of laxity. One to 2 cm. of shortening of the extremity is not an uncommon finding. All the soft tissue elements on the medial side of the joint exhibit varying degrees of laxity, and occasionally the abnormal stress and strain inflicted upon the soft tissues may result in formation of an effusion in the knee joint and produce pain.

Treatment

As previously noted, spontaneous correction may occur just as in other forms of osteochondroses. This response is not governed by any conservative measures of treatment. However, it is important to try to prevent undue strain on the knee joint and to correct existing pronation of the feet by braces and corrective shoes. In mild forms such measures should be continued until there is clinical and roentgenographic evidence that the deformity is stationary.

In adolescence, however, if the deformity is severe and dysfunction is great, surgical intervention is justifiable. Many cases disclose that the deformity does not increase when the epiphysial plate is closed. Hence, in adolescence in which there is obvious damage to the plate, it is advisable to postpone any operative measure until closure of the plate is achieved. In the adolescent group, if surgical correction is properly carried out, no recurrence is a certainty.

In the infantile group, management is more difficult because of the element of growth. Not infrequently the lesion progresses for three or four years and then becomes stationary without becoming reactivated at a later period. However, in others, after a stationary period, the deformity continues to increase in severity. Even in the infantile group the deformity may be of such nature that surgical intervention is desirable. It is important to execute corrective surgical measures only in the stationary period, because, if the process remains stationary, no recurrence will ensue. However, if the process should become reactivated, recurrence of the deformity is a certainty.

It is not the purpose of this presentation to outline the various methods of surgical management. However, it should be mentioned that an excellent approach to this problem is the open-wedge osteotomy at the site of maximum deformity on the medial side of the leg. The osteotomy should be done immediately below the epiphysis at the apex of the angulation. By this method, angulation is corrected and some length of the tibia is regained. At this time any existent internal torsion of the tibia is also corrected. The limb should be slightly overcorrected and encased in a plaster spica from the costal cage to the toes with the knee slightly flexed.

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BOWLEGS AND KNOCK KNEES IN CHILDHOOD

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Bowlegs and knock knees are common expressions denoting certain deformities of the lower extremities. They are merely anatomic terms, however, and the deformities may be caused by many etiologic factors. Every physician who treats children realizes the prevalence of these deformities and is perplexed by a lack of satisfactory classification and subsequently a successful therapeutic program. A great deal has been written about the subject, but confusion still exists as to the physiology and pathology.

ETIOLOGY

During the early part of the century almost all cases of bowlegs and knock knees were thought to be due to rickets. With the advance of physiologic chemistry and a more thorough understanding of bone metabolism and vitamin therapy, this cause of these deformities has been largely eliminated. Strange as it may seem, the problem is still present with perplexing frequency. It is now known that the diagnosis of rickets previously attached to many of the deformities was incorrect.

A large number of these deformities have been found to be physiologic variations from the normal. Growth is extremely rapid at the proximal tibial and distal femoral epiphyses of the growing child. Weight bearing and certain other unknown extrinsic factors may cause unequal growth of these two growth centers, producing either knock knees or bowlegs associated with torsional changes. There is no pathologic condition involving the bones, joints or epiphyses, and roentgenograms are normal in appearance. Deformities are symmetrical, with equal contours in either extremity. There is no disturbance in the

metabolism of calcium or phosphorus. The deformities are not static, and in time correction occurs spontaneously in almost all cases. The administration of vitamin D does not hasten or alter in any way the improvement which these children show. In fact, the administration of too large a quantity of these preparations may result in a vitamin A intoxication, because practically all vitamin D preparations contain varying quantities of vitamin A (Fig. 77).

Fig. 77. Roentgenogram showing bone changes occurring as result of hypervitaminosis A in a normal child receiving a triple normal dose of Oleum Percomorphum over a period of 1 year.

Frequently these deformities will shift, the extremities swinging as a pendulum from bowleg to knock knee in the course of two or three years without any form of treatment. In those cases of knock knees and bowlegs which are moderately excessive it is necessary to place a small wedge on the inner aspect of the heel to protect the foot. In mild to moderate forms of this condition there is also a minimal degree of torsion of the tibia. This physiologic change in the rapidly growing extremity is of utmost importance and should be recognized as a common cause of bowlegs and knock knees which needs no specific treatment other than the reassurance of the parents, and observation and protection of the foot.

CLASSIFICATION

With the foregoing group of physiologic deformities in mind, it is now possible to propose a simplified classification, as follows:

I. PHYSIOLOGIC
II. PATHOLOGIC
  A. Ligamentous abnormalities
    1. Congenital absence or laxity
    2. Injury
  B. Torsional or angular changes of the femur or tibia, or both
    1. Congenital developmental defects
    2. Vitamin deficiency
      a. Rickets

b. Vitamin-resistant rickets
3. Disease
   a. Blount's disease
   b. Fanconi syndrome
   c. Renal rickets
   d. Chondro-osseous dysplasia
   e. Fibrous dysplasia
   f. Osteomyelitis
4. Injury
   a. Trauma to the epiphyses
   b. Trauma to the shaft
   c. Surgical trauma
   d. X-ray therapy
5. Paralytic

LIGAMENTOUS ABNORMALITY

Although anomalies of the ligaments about the knee in children are infrequent, there may be complete absence, relaxation or attenuation.

a b

Fig. 78. Congenital anomalous development of collateral ligaments of knee. Standing with very little weight (*a*) and with full weight on leg (*b*).

When they occur, usually more than one ligament is involved, and the knee is quite unstable. The condition may be noted prior to weight bearing, but, if not severe, it is usually recognized when the child begins to walk (Fig. 78).

Injuries occur rarely and present no problem as far as diagnosis is concerned, with the history of an accident and the clinical examination

The *treatment* of these ligamentous conditions should be repair as soon after injury as possible. In those cases due to congenital deformity or in cases seen late after injury, various reconstructive measures should be utilized.

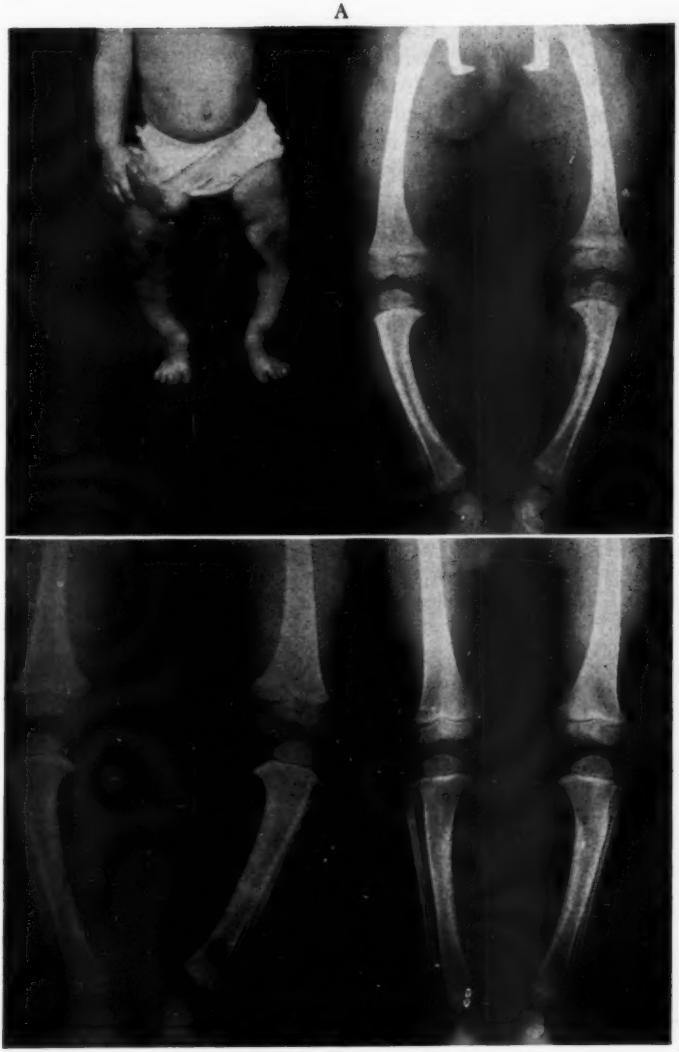
CONGENITAL DEVELOPMENTAL DEFECTS

It is generally recognized now that the majority of bowleg and knock knee deformities are due to torsional changes in the femur or tibia, or both (Fig. 79), because of congenital positions upon which are usually exerted developmental forces of certain sitting and sleeping habits. Bohm[5] first described the developmental changes of the lower extremities during early growth from bowleg to knock knee and back again with final spontaneous correction. He felt that in certain instances these changes might not occur, owing to some atavistic reversion or developmental arrest, and the child retain the deformity (Fig. 80). This was later emphasized in papers by Nachlas[12] and Blount.[2] Fitzhugh[7] called attention to sleeping and sitting habits and play postures as factors that might enhance development of these deformities and in some instances possibly might be the direct cause.

Recently, Kite[10] and Knight[11] emphasized the necessity of recognizing these so-called congenital and developmental torsional deformities early and instituting proper therapy. Howorth,[8] in discussing these papers, felt that these deformities should certainly be recognized and treated, but stated that while we have good anatomic and descriptive findings of these conditions in infants and children, we are completely "at sea" as to the specific etiology of these deformities.

We do have this large group of deformities, usually mild, but present. We know that a large group of them are variations of normal or the normal changes of development, and regardless of what is done, the end result will be good; however, there are those that persist. Since a number of persons reach adulthood with definite pathologic knock knee or bowleg deformity, it is our duty to try to determine the causes as early as possible and to correct them before adulthood is reached. There has been a widespread opinion that all torsional deformities in childhood will spontaneously correct. A study of 1500 school children in Los Angeles, however, tended to refute this opinion.[9] Eight to 10 per cent of these children showed abnormal torsional changes in the teen-age groups. Whitman[14] found 400 cases of abnormal bowlegs and 32 cases of knock knee among 2000 adult males. He and numerous other observers have concluded that natural forces are often ineffective in completing cures. We do not know which will persist, and therefore it is best to recognize the condition early and treat it then, since treatment in the early stages is simple.

A



B

Fig. 79. A, Child with congenital internal torsion of tibias, enhanced by sleeping in knee-chest position. B, Roentgenograms showing tendency toward correction of congenital, developmental torsion over period of 1 year. Note normal epiphyses and epiphysial plates and bone architecture.

The degree of deformity in the early stages does not denote the outcome. Sometimes those that are the most severe correct themselves spontaneously (Fig. 81), while others that seemed minimal or moderate persist as abnormal structural changes into adult life. Sleeping and sitting positions maintained by infants may not act as primary deforming factors, but they most certainly contribute to the continuance and aggravation of deformity. Practically all children with excessive internal rotational or bowleg deformities are face sleepers or knee-chest sleepers who hold the feet inverted. Those with eversion deformities with knock knee are generally back sleepers or face sleepers

Fig. 80. Valgus knees as result of congenital developmental changes which did not make shift back to straight position.

who hold their feet and legs in an everted position. If the infant is taught from the start to tolerate side sleeping or frequent changes of position, none of these fixed torsion-producing difficulties is encountered. Later in infancy the waking habits tend to follow the sleeping position. Among knee-chest sleepers the child with internal torsion tends to sit on the feet (Fig. 82) and often has, in addition to the internal torsion and bowlegs, calluses over the heads of the talus and lateral calcaneus called "television bumps"[11] by some. The everter sits in the tailor position or with the knees flexed and the feet everted. The "infant walker" often offends as the child propels this vehicle with the feet everted and the knees and hips flexed.

The *treatment* of this group of deformities should begin as soon as they are recognized by advising side sleeping or frequent changes of sleeping position in infants. If the fixed sleeping position has already

developed, it is almost impossible to expect the parents to break the child of this habit without help. One of the best methods of doing this is by the use of a modified Denis Browne splint. This is tolerated well in children under three years, but rarely after this age. In the case of bowlegs, the long cross bar is used and the feet are gradually rotated outward to a position of some 50 to 60 degrees. In the case of knock knees, a short cross bar is used and the feet are gradually rotated inward some 45 degrees. If the child does not tolerate this, it is usually because of an attempt to rotate too rapidly, ill fitting shoes or unco-

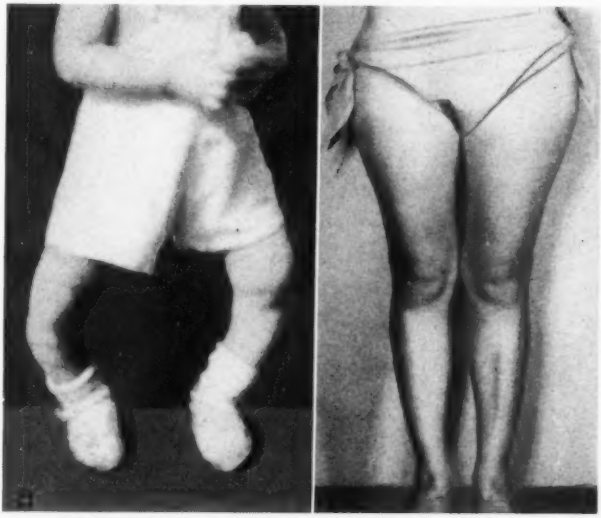


Fig. 81. *a*, Child 18 months old with severe internal torsion of tibiae. *b*, Same child 15 years old after normal development and no treatment whatsoever.

operative parents. Sometimes one leg may rotate inward while the other rotates outward, as the deforming positions. In these instances the bar can be altered accordingly.

Torsional stretching exercises, while theoretically good, have generally proved unsuccessful. Frequently the mother performs these for a short time and then soon stops. Long leg braces have generally been found to be unnecessary and are usually of little value. Shoe corrections are of no benefit so far as the correction of the deformities of the legs is concerned and should be used only to protect the arch in case the deformity is severe enough to cause pronation of the foot. Because of the line of weight bearing, pronated feet occur secondary to severe bowlegs and knock knees; therefore the corrections should be in the form of inner heel wedges or possibly arch supports for both deform-

ities. When the corrective shoes are used, the parents should be warned that the child may turn in even more than when barefooted.

Some children do not respond to the previously described conservative measures, while others are seen too late for these measures to be effective. Surgical correction is indicated for this group, and should be done in those who are six years of age or more when the deformity is

Fig. 82. Sitting posture spontaneously assumed by child with congenital, developmental internal torsion who also sleeps in knee-chest position.

cosmetically objectionable. The procedure of choice for this condition is that of an osteotomy, usually performed at the proximal tibial diaphysis. It is to be definitely understood that all cases of such deformities should be thoroughly worked up with an accurate history and physical examination, x-ray and laboratory examinations and a period of observation.

In the foregoing group of cases laboratory studies should prove to be perfectly normal and roentgenograms show no abnormal changes other than the general structural varus or valgus of the tibia or femur, or both.

VITAMIN D DEFICIENCY DISEASES

Vitamin D deficiency will produce disturbance of growth of the epiphyses and, therefore, deformities of bowlegs and knock knees. This is a systemic condition, and its clinical and x-ray manifestations are well known (Fig. 83). Blood and urine chemistries reveal the typical changes in the calcium-phosphorus metabolism. Rickets is definitely not common in the United States now. The condition should be diagnostically differentiated by roentgenogram from such similar lesions as

Fig. 83. Child with true rickets, who has had no supplementary vitamin D since birth. Typical x-ray changes.

vitamin-resistant rickets, the Fanconi syndrome or renal rickets. These conditions will be further discussed later in this paper. Early treatment with proper vitamin D will usually result in regression of these deformities. Those deformities that do not regress may require osteotomies later.

Occasionally we see children with all the typical findings and features of rickets, yet with the history of adequate vitamin D therapy (Fig. 84). In these cases it is characteristic that large doses of vitamin D are required to bring about the excretion of calcium in the urine and changes in the blood chemistry, along with roentgenographic evidence of healing. The vitamin dosages required in these cases may range as high as 500,000 to 1,000,000 units of vitamin D (without A) daily. This condition was first described by Albright,[1] who called it vitamin-resistant rickets. The deformities associated are usually severe. Dwarfism

is a feature in most instances. McCarroll and Pederson[13] found that vitamin D-resistant rickets was the most common cause of dwarfism in their clinic.

Accurate classification and diagnosis can be made by history, examination, a few blood studies and urinalysis. The most important of

a

b

Fig. 84. *a*, Six year old child, receiving usual supplementary vitamin D since birth. Dwarfism. Rachitic changes by x-ray. *b*, Two years later after receiving 300,000 units of vitamin D daily.

these are determinations of serum calcium, phosphorus and alkaline phosphatase. There is a strong familial tendency. It must be differentiated from renal acidosis and the Fanconi syndrome, both of which may produce similar x-ray and blood chemistry changes, by signs of kidney disease and absence of disturbed plasma electrolytes. All degrees of severity are seen. The deformities may be mild in the early stages, but most of them are severe. Frequently bowing is seen in one extremity, while knock knee is present in the opposite one.

Improvement may be expected with vitamin D therapy. This is manifested by spontaneous tendency toward correction of deformity and roentgenographic signs of healing of the epiphyses. The initial dosage of vitamin D is empirical. The blood and urine studies should be checked carefully and a certain dosage begun. We usually start with 25,000 units and watch the Sulkowitch test daily and either increase or decrease the dosage accordingly. If the Sulkowitch test can be maintained at 1 or 2 plus, healing can be expected without too great danger of hypercalcinuria. It is best to work out the proper dosage in the hospital, and when this is determined, it may be maintained on an outpatient basis. Usually the parent can be taught to do the Sulkowitch test, which is performed at home daily. Blood studies can be performed once monthly. There is a fine dividing line in the dosage of vitamin D between that which is adequate to control the rickets and that which may cause hypercalcinuria and kidney damage. Once the healing begins, the amount of vitamin necessary to maintain calcium and phosphorus balance may drop, and the dosage must be reduced. A low serum phosphorus may continue even though the Sulkowitch runs 1 to 2 plus, and roentgenograms show continued healing.

If, after a period of adequate therapy, functionally or cosmetically disabling deformities persist, then osteotomies should be performed. *When such operative procedures are done, those patients being treated with vitamin D might exhibit hypercalcemia and hypercalcinuria.* It is necessary, therefore, that the vitamin therapy be discontinued just prior to operation and the blood and urine calcium and phosphorus values observed closely. Vitamin D is then resumed as indicated by chemical studies after operation. Usually the full dosage may be resumed two weeks postoperatively. Recurrences of rachitic activity and deformity are likely if therapy is interrupted, and frequently therapy must be continued until growth is complete, and sometimes even into adult life.

BLOUNT'S DISEASE (OSTEOCHONDROSIS DEFORMANS TIBIAE)

One of the first conditions producing bowlegs that was separated from rachitic deformities was osteochondrosis deformans tibiae (Fig. 85). This condition was first described by Erlacher[6] in 1922 and emphasized by Blount[3] in 1937, when he reviewed all previously reported cases and added some of his own. His work on pathology of this condition and its correction has added greatly to our understanding. He suggested the former name, but it is probably best known now as Blount's disease. The deformity may be unilateral or bilateral, mild or severe. It is usually not noted until weight bearing is begun, and often is not evident until several years after birth. It is usually pro-

gressive. Laboratory studies are normal; the x-ray findings make the diagnosis, and show tibia vara with abrupt angulation just below the proximal tibial epiphysis. The epiphysial line is sometimes irregular and is associated with a prominent beaklike, recurving medial metaphysis.

Conservative *treatment* of this condition is not effective. Surgical correction can be obtained by performing an osteotomy at the proximal

Fig. 85. *a*, Blount's disease, moderate. *b*, Blount's disease, severe.

tibial diaphysis. There are many types of osteotomies, any one of which will produce the desired result. As a rule, osteotomy is delayed until growth is almost complete in order to prevent recurrence. If the deformity is severe, osteotomy should be done in early childhood in order that secondary changes such as attenuated ligaments and pronated feet may be prevented. When operation is performed this early, it should be expected that the deformity will recur and a second osteotomy would be necessary. Correction attained at the cessation of growth will persist.

In 1949 Blount[4] described epiphysial stapling which can be used to

slow down growth of the lateral side of the epiphysial plate and thereby result in a gradual progressive correction of the deformity. This also may have to be performed more than once if used early. This procedure is quite effective for correction of this condition.

RENAL RICKETS AND FANCONI SYNDROME

Renal rickets due to tubular disease or the Fanconi syndrome (Fig. 86) may cause severe disturbances in epiphysial growth with deform-

Fig. 86. Fanconi syndrome. Note the severe deformities; pseudofractures in ulna and radius; metaphysial changes.

ities. These conditions will show disturbances in kidney function and in plasma electrolytes. The roentgenograms usually show osteoporosis of severe degree along with rachitic epiphysial changes. So-called pseudofractures of the long bones and ribs are not uncommon. The prognosis in these conditions is poor, and treatment in general is of no avail.

CHONDRO-OSSEOUS DYSPLASIA AND FIBROUS DYSPLASIA

When such affections of bone as chondro-osseous dysplasia (Ollier's disease) (Fig. 87) or fibrous dysplasia involve the femur or tibia, one

Fig. 87. Chondro-osseous dysplasia with involvement of medial epiphysis of lower tibia; leg length discrepancy.

may expect deformities to occur. The bowleg or knock knee or other deformity may occur because of abnormal growth of the epiphysis, which has been disturbed by one of these conditions. When such elements as fibrous tissue or cartilage, or a combination of both, have replaced or are replacing bone, curvatures may develop gradually as the result of gravitational and muscular stresses which ordinarily normal bone would be strong enough to resist. Frequently these conditions will also produce inequality in growth of extremity length. These conditions show characteristic changes by roentgenogram and are readily recognized by competent roentgenologists.

Treatment may be somewhat complicated. At times when the lesion

is discovered early and the deformity is gradually increasing, it may be necessary to discontinue weight bearing. The most satisfactory method of accomplishing this in children is by the use of an ischial weight-bearing brace. It may be necessary to protect the extremity by the use of such an appliance for several years. Usually after the extent of the lesion has been decided, definitive surgery may be accomplished. Frequently an osteotomy is necessary to correct the deformity, and one may have to perform this more than once until growth is complete. In fibrous dysplasia the lesion may have to be eradicated completely by surgery, and the remaining defect filled in with grafted bone. In those instances of accompanying inequality of leg length growth, the discrepancy should also be followed by x-ray growth studies and bone age determinations so that epiphysial arrests may be performed at the proper time to give equality of leg length when the termination of the growth period has been reached.

OSTEOMYELITIS

Infections involving the epiphysial plate may produce delay or cessation of growth. Sometimes when the disease is in the metaphysis and the epiphysis is uninvolved, the hyperemia caused by the inflammatory reaction may stimulate growth. Fortunately, infections rarely pass the epiphysial barrier, and cessation or delayed growth of the epiphysis is rare.

The infection must be treated by the usual medical and surgical methods indicated by the type of infection present. If growth disturbances and deformity occur, the case is followed and treated as described under injuries causing similar difficulties (see Injury).

INJURY

Injuries which involve the epiphysial cartilage plate may produce cessation or delay in growth. If this should occur on only one side of an epiphysis, deformity will occur, resulting in bowleg if the involvement is on the medial side and knock knee if the damage is on the lateral side. Here again, disturbance in leg length will occur, and this should be borne in mind in following up and treating these cases.

The *treatment* of the deformity must necessarily be surgical. This may take the form of correction by osteotomy in the metaphysial area of the epiphysis involved or by use of staples to arrest temporarily the growth of the opposite epiphysis (see Blount's Disease). Should injury or infection involve the metaphysis of a bone near the epiphysis, there may occur a temporary increase in the rate of growth of the epiphysis in juxtaposition with increasing deformity. These should be watched

closely. Sometimes there may be a tendency to spontaneous correction, but if not, staples may be used effectively to temporarily slow down the growth of the epiphysis on the same side until the deformity corrects, or osteotomy may be used to correct the deformity.

As a rule, these deformities are never of the same severity or import as those due to epiphysial arrests. Rarely is leg length inequality any problem in these cases. Angulatory changes occur with malunited fractures. In the very young children these will tend to remodel and correct themselves to a large extent and often completely. In the older age groups, if the deformity is cosmetically or functionally bad, it should be corrected by osteotomy.

All these deformities due to injury or inflammation will have a history of such involvement and are rarely of diagnostic difficulty. Sometimes the injury at the time may have seemed inconsequential and may have been forgotten, such as a fall from a height with compression of the lateral epiphysis in a young child with subsequent development of knock knee deformity without pain except for a few days after the injury.

SURGERY

While on the subject of injury, it should be mentioned that unfortunately surgcial procedures at times may injure one side of an epiphysis, and therefore produce progressive deformity (Fig. 88). To be

Fig. 88. This patient had osteochondroma of the lateral side of the lower femur, removed 3 years previously. The lateral femoral epiphysis is damaged. Progressive valgus deformity.

sure, the majority of such cases are due to poorly planned surgery. Any time surgical intervention is necessary in an area near a growing epiphysis, this should be borne in mind and the epiphysis carefully avoided.

It may be well to mention at this point that it is disturbance of the peripheral part of an epiphysial plate that is most likely to cause interference with growth. The central part of the epiphysial plate may be disturbed without causing any interference with growth.

X-RAY

X-ray therapy used about or near an epiphysis in small children may cause a disturbance in epiphysial growth. Sometimes it may cause cessation of or delay in growth of the entire epiphysis with decrease in leg length of the extremity. In rare instances which we have seen, one side of an epiphysis has been delayed while the other seemed to grow at a normal rate, causing the anatomic deformity of bowleg or knock knee. X-ray therapy near growth centers should be used with extreme caution.

Fig. 89. Knock knee deformity in a patient after poliomyelitis. Note the low left pelvis, abducted hip, external torsion of the tibia, and varus of foot, all due to myostatic contracture of the iliotibial band.

PARALYTIC DEFORMITIES

We often see bowleg and knock knee deformities associated with paralytic conditions of the lower extremities. As a rule the reason for the paralysis is inconsequential, and the same deformity may be due to numerous reasons; unequal muscle pull, position, gravitational forces and improper bracing all may tend to produce these deformities in a growing child with paralytic lower extremities. For the most part, if the child is seen early, deformities can be prevented, for we are able now to anticipate most of these occurrences by the knowledge of the muscle picture in each individual case.

Valgus deformities of the tibia are most frequently encountered.

Sometimes one contracted structure may cause numerous deformities. For example, myostatic contracture of the iliotibial band will cause flexion, abduction deformity of the hip, scoliosis, valgus and flexion deformity of the knee, external torsion of the tibia and often varus of the foot (Fig. 89). This is only one example of many that might be mentioned.

Prevention by proper bracing, stretching and positioning is the *treatment* of choice. When present, the deformities are usually corrected by osteotomy and the extremities then braced properly to prevent recurrence.

SUMMARY

The recognition of bowleg and knock knee deformities is the concern of every physician who treats children, and every effort should be made to institute a proper therapy program as early as possible.

Although numerous controversies exist regarding the etiology of bowlegs and knock knees, a simplified and workable classification of these deformities is presented. Every case of bowleg or knock knee deformity should be studied in order to classify it properly and to institute a sound therapeutic program. Observation should be an important part of almost every therapy plan.

Many different pathologic conditions involving growing bone may cause bowleg and knock knee; these have been discussed.

Ligamentous instability due to injury or anomalous development may rarely cause deformity.

Physiologic changes in the alignment of children's legs either in the form of bowlegs or knock knees should be recognized and not confused with the pathologic deformities. Vitamin D therapy in these cases is not indicated and may be harmful if excessive.

Rickets, once the common cause of bowleg and knock knee deformities, has been largely eliminated, although vitamin D-resistive rickets must always be considered a possible etiologic factor.

Congenital developmental defects are now considered to be the main cause of bowleg and knock knee deformities. Although this is a controversial group, treatment is simple if started early, and a good result may be expected.

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9-11 Medical Court
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TRANSIENT SYNOVITIS OF THE HIP JOINT

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This clinical entity has been variously called transient synovitis, phantom hip, toxic synovitis, transitory synovitis, intermittent hydrarthrosis of the hip, coxitis serosa, transitory arthritis. We feel that the term "transient synovitis" best describes the true nature of the condition. The synovium of the hip joint appears to be the principal site of involvement. The involvement is transient as compared with synovitis caused by a specific and known etiology.

This report is based upon 52 consecutive cases treated on the orthopedic services in Children's Hospital, Pittsburgh, Pennsylvania.

ETIOLOGY

The literature is replete with possible etiologic factors. These include "toxic factors" such as chronically diseased tonsils and upper respiratory infections. Some authors have postulated an allergic basis. Trauma has been recorded in some instances. No one etiology appears to satisfy in all cases. In many the etiology is not discernible.

INCIDENCE

Transient synovitis of the hip joint is almost exclusively a childhood disease occurring between three and 10 years of age; males are more commonly affected. It is usually characterized by an acute onset of pain in the hip, and/or limp. In our experience it has become one of the most commonly recognized causes of pain in the hip in this age group.

The average age of the patient at the onset of symptoms was 5.9 years; the youngest was two years, and the oldest in the series was 13½ years of age.

Eighty per cent of the cases were male. This figure compares favor-

ably with other published series. In some the predominance of the male incidence reached 90 per cent.

Racial incidence in the past was never significant. In this group over 90 per cent of the patients were white. This represents a higher incidence than the ratio of white to colored admissions to the Children's Hospital. The numerical variation is not conclusive.

SYMPTOMS AND SIGNS

Eighty-eight per cent of the patients complained of pain and limp. Twelve per cent had a limp only. Of those who complained of pain, 55 per cent had pain in the anterior hip region. Twenty per cent were recorded as having pain in the knee only. The remainder complained of diffuse pain in the leg, usually confined to the thigh, and seldom below the knee.

The right hip was involved in 60 per cent of our cases. The predominance of one side over the other has no known significance.

The majority of the patients were seen within the first or second day after the onset of symptoms. Eight cases were not seen until between one and three months after symptoms had occurred. The average duration of symptoms prior to admission was 8.7 days.

There was either a recent or concomitant upper respiratory infection or chronic infection in the tonsillar tissue in one fifth of the patients. Only one patient had a definite allergic history. Four patients had a history of apparently related trauma.

On physical examination all patients showed some definite restriction of hip motion, and in the majority there was a hip flexion contracture and restriction of rotation and abduction. The degree of restriction of motion varied considerably. In all cases there was some demonstrable hip motion. Tenderness over the hip joint anteriorly was not a consistently recorded finding. Tenderness over the greater trochanter was almost never found. Palpable soft tissue thickening in either of these areas was not seen. Inguinal adenopathy was not an associated physical finding. Bilateral involvement was not seen. Atrophy of the thigh was not demonstrable in early cases. There was no associated involvement of other joints. Elevation of the systemic temperature was found, but it seldom went above 101° F. None of the patients in this series ran a septic type of fever. Elevation of temperature, if present, usually subsided within a day or two without antibiotic therapy. Salicylates were used only as necessary for the control of pain.

ROENTGENOGRAPHIC EXAMINATION

X-ray studies in all instances were negative for alterations in the bony structure of the hip joint. Relative osteoporosis was not found.

Our series excludes all who had bony changes consistent with true Perthes' disease, e.g., sclerosis of the epiphysis or radiolucent area along the metaphysial edge of the epiphysial plate.

In 60 per cent of the cases the roentgenograms showed definite soft tissue swelling of the capsule of the hip joint (Fig. 90). The soft tissue

A

B

Fig. 90. *A*, Roentgenogram showing distended capsular soft tissue shadows of the left hip. This also shows obturator internus soft tissue swelling. *B*, Retouched photograph showing the soft tissue shadows outlined.

shadow of the capsule is thickened and bulges laterally as compared with the uninvolved side. In the remainder of the cases the x-ray picture was either mildly suggestive or negative. Soft tissue swelling of muscle groups about the hip joint (obturator internus, gluteus

A

B

Fig. 91. Follow-up film taken 6 weeks later. *A*, Roentgenogram showing symmetrical capsular soft tissue shadows. *B*, Retouched photograph with capsular soft tissue shadows outlined.

minimus, iliopsoas) was not nearly as consistent a finding and not as easily interpreted as the capsular swelling.

Subsequent follow-up x-ray films have failed to reveal changes in the bony structure. Soft tissue changes recede to normal (Fig. 91).

LABORATORY FINDINGS

The laboratory studies were of help chiefly in a negative manner. The white blood cell count was usually within normal limits. In only eight cases were white blood cell counts higher than 10,000. The differential count showed no consistent shift. Febrile agglutinins were done in two cases and in both instances were negative. Tuberculin testing with old tuberculin was consistently negative in dilution down to 1:100. Serologic examination for syphilis was also negative.

The sedimentation rate was usually normal and, when elevated, showed no constant relation to the acuteness of the case, the temperature elevation, the white blood cell count or its differential. The highest sedimentation rate recorded was 38 mm. per hour. In those cases with an associated upper respiratory infection the sedimentation rate varied from 2 to 30 mm. per hour, the majority being within the variation of normal.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of this condition is not usually difficult when a careful physical examination is performed and when adequate roentgenograms for both bone and soft tissue detail are taken. Established Perthes' disease is most readily distinguished by the roentgen changes. Septic arthritis is characterized by an acute, febrile clinical picture, and usually there is complete restriction of hip motion. Occasionally aspiration of the hip joint will be necessary for differential diagnosis. The finding of pus or a subsequent positive culture would, of course, indicate the true diagnosis.

The more chronic nature of tuberculous synovitis, the subsequent x-ray changes, the associated characteristic laboratory findings and positive tuberculin, and chest x-ray findings should point the way to the proper diagnosis. Aspiration for culture and smear may again be diagnostic. Hematogenous osteomyelitis prior to definite bone changes can usually be distinguished by the septic nature of the patient. Blood culture should be performed when hematogenous osteomyelitis is considered. Rheumatic fever and monarticular early rheumatoid arthritis will be differentiated by the course of the disease. In the older age group, slipped capital femoral epiphysis must also be considered and, again, the characteristic x-ray findings will separate these from transient

synovitis. If the symptoms and signs do not respond rapidly to therapy, the diagnosis should be seriously questioned.

TREATMENT

Treatment in this series consisted in complete bed rest with split Russell traction applied to the affected extremity. Three to 8 pounds of weight were used, depending upon the size of the patient. Traction was continued until all signs of muscle spasm had disappeared, and the patient had a completely normal range of painless motion re-established. Pain usually subsided within 24 to 48 hours, and motion would return to normal within three to four days. Approximately 40 per cent of the patients were then placed in a single hip spica cast and ambulated with the aid of crutches. For six weeks the hip spica cast was used to assure nonweight bearing. Protection from weight bearing is thought desirable to prevent recurrence and to protect the hip if, for example, it should be an unrecognized case of Perthes' disease. There has been no instance of recurrence of the symptoms upon graded ambulation, and a normal range of hip motion was readily re-established. Subsequent x-ray studies have always been negative.

The remaining 60 per cent of the patients were kept at complete bed rest for one to two weeks longer, traction having been discontinued. The patients were then ambulated. Only one patient in this group had sufficient persistence of symptoms to warrant further therapy. This patient was readmitted and, after a further period of traction and bed rest, had complete and lasting relief of symptoms. Follow-up x-ray studies have not shown any untoward changes. Review of the clinical course in the hospital would suggest that therapy had been discontinued too soon.

It would seem unnecessary to immobilize these patients in hip spica casts if the criteria for discontinuing traction and complete bed rest are carefully followed. Cooperation of the family upon discharge from the hospital is most important. Nonweight bearing and gradual ambulation in the home are only as successful as the cooperation obtained from the family.

The author shares the opinion that transient synovitis may be the first stage of Perthes' disease. If this is true, early recognition of this condition and adequate therapy while the vascular embarrassment in the synovium and hip joint is still reversible should reduce the incidence of Perthes' disease.

The relationship to Perthes' disease is further strengthened by the usual age of the patient, the male sex predominance, and the similarity of the clinical picture except for the duration of symptoms. The soft tissue x-ray findings are similar to that seen in early Perthes' disease,

and only the bone changes differentiate them. The laboratory studies are equally negative.

CASE HISTORY

L. K., a 6 year old white boy, had a history of pain in the left hip, anterior aspect. A limp developed shortly after the onset of the pain. The symptoms had been present for 3 days prior to admission. There was no history of trauma or upper respiratory infection. His past medical history was noncontributory, and was negative for allergy. The patient had been in good health until the onset of symptoms.

Physical examination was essentially negative except for the left hip. There was no elevation of the temperature. The right hip had a free, painless, normal range of motion. The left hip had a flexion contracture of 30 degrees, with 60 degrees of further flexion possible to 90 degrees. Internal rotation was 20 degrees; external rotation, 30 degrees; abduction, 30 degrees. No tenderness was elicited over the anterior aspect of the hip joint. There was no significant adenopathy.

Laboratory studies were as follows: white blood cell count, 8600; differential count —48 polymorphonuclear leukocytes, 52 lymphocytes; sedimentation rate, 4. Urinalysis was negative.

Roentgenograms showed the typical capsular soft tissue swelling. The bony structure was normal.

Treatment consisted in complete bed rest and split Russell traction. The pain subsided in 24 hours. Normal motion returned in 4 days. The patient remained in the hospital 17 days. He was ambulatory at discharge and was weight bearing and symptom-free.

Follow-up roentgenograms showed no alteration in bony architecture, and the capsular swelling was gone. The patient has remained symptom-free, and there is a normal range of hip motion.

CONCLUSIONS

1. Transient synovitis of the hip joint is a definite clinical entity.
2. Its etiology is probably varied and for the most part obscure.
3. Toxic or allergic hypersensitivity to a focus of infection cannot be substantiated.
4. There is a low incidence of associated trauma.
5. Transient synovitis of the hip responds to nonweight-bearing therapy.
6. Transient synovitis may represent the first and reversible stage of Perthes' disease.

The author wishes to express his thanks to Drs. John S. Donaldson, Harold H. Sankey, Carl J. Pessolano and Albert B. Ferguson, Jr., for permission to include their cases in this report.

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CONGENITAL DYSPLASIA OF THE HIP

Recognition and Treatment

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In congenital dislocation of the hip prompt diagnosis and early institution of treatment are of utmost importance. It has been repeatedly shown that excellent results can usually be anticipated when treatment is instituted before six months of age. The authors are not aware of a single patient who "outgrew" the condition without treatment. If treatment is delayed until the child is three years of age or older, certain adaptive changes occur, not only in the articulation, but also in the surrounding soft tissues. These changes preclude restoration of the hip to a normal condition, and the joint is practically always destined for premature degenerative changes. Such arthritic changes occur at or before the climax of the person's social and economic productiveness and, despite reconstructive measures, are a source of permanent disability.

Some improvement in results in the treatment of congenital dysplasia of the hip can be anticipated by the refinement of present techniques of diagnosis and treatment, and the introduction of new ones from time to time. At present, however, the chief problem in this respect is the creation of an increased awareness of this condition among pediatricians and practitioners caring for "well babies," so that children thus affected can be referred for treatment before irreversible secondary changes supervene.

The purpose of this paper is to review the incidence, classification,

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pathologic anatomy, and clinical and roentgenographic diagnosis of dysplasia of the hip. Variations in treatment will be discussed briefly. Certain significant physical findings will be discussed in detail. Roentgenographic examination of the pelvis, including both hips, is necessary before a definite diagnosis can be established.

INCIDENCE

Perhaps the most distinctive single feature in the occurrence of dysplasia of the hip is its higher incidence in girls, the ratio being given as between six and 10 to one by various authors. Inheritance of dislocation of the hip has been repeatedly observed, Putti[22] reporting it as high as 13 per cent in 1900 cases. Jansen[13] noted that it may alternate with other abnormalities in siblings. In certain sections of the world there is apparently a higher incidence, particularly in northern Italy, southern France and Czechoslovakia, where it may be seen as frequently as four cases per 1000 population. Environmental factors undoubtedly add to the higher incidence in these areas because of the age-old custom of binding the legs together during infancy. Shands[23] noted that dysplasia of the hip occurs more frequently in the northern areas of the United States and that among Negroes it is seldom observed.

HISTORY

In the days of Hippocrates the disabling effects of dysplasia of the hip were recognized. The inequality of leg lengths, the characteristic limp, the pelvic tilt and the tendency to bear weight on the uninvolved side with a flexion contracture of the affected hip were all described. Manipulations were done on those cases recognized in childhood, and powerful windlasses and overhead suspension traction methods were used to reduce the dislocation in adult cases.[1] It was not until the nineteenth century that Paletta[19] and Dupuytren[7] described the detailed pathologic anatomy of congenital dislocation of the hip. The latter described the clinical findings as well, and expressed the commonly held attitude of his day as to its prognosis by stating that "the deformity is incurable." Since then, the recognition of other associated clinical findings has added considerably to our understanding of this condition.[6, 9, 10, 18, 22] It was found that developmental defects often occur in the "normal hip" of the opposite side. The clarification of fetal and neonatal causes and the differentiation among dysplasia, dislocation, subluxation and preluxation[21, 22] have been made more distinct.

The concept of gradual closed reduction and the formation of a normal hip by maintaining this reduction in various attitudes for long periods of time was taught by the Vienna school. In Italy the first successful open reduction of a dislocated hip was done in 1888. Because of failures of treatment by closed reduction, American surgeons began to prefer the operative method. The indications for closed and open reductions have been clarified. The conservative treatment, by abducting the hips, has progressed from cumbersome strapping of the legs to a large board to the convenient Frejka pillow splint and the novel "guided abduction" of Craig.[4] When indicated, procedures now used consist in open reduction with removal of the obstruction to the relocation of the femoral head[17, 24] and, for the more severe cases, the formation of a more effective superior margin of the acetabulum by the

shelf operation and correction of the valgus and rotational deformities of the femoral neck by the Zahradnicek operation and by derotational osteotomy.[24]

CLASSIFICATION

Clinical Classification

Most of the earlier descriptions of dysplasia of the hip were based upon the factors of etiology, with particular attempts to separate that phase in embryonic, fetal or neonatal development during which the causative factors disrupted the normal chain of events. A more useful classification, based on the clinical manifestations, was offered by Hass[10] in 1951.

Typical Cases. This group, representing over 90 per cent of the cases, is characterized by a markedly high incidence in girls and a definite hereditary influence in as many as 25 per cent of the cases. A partially developed subluxation or "preluxation" (Putti)[21, 22] is present in the joint and may progress to subluxation or frank dislocation. In the fully developed dislocation the femoral head migrates posteriorly and superiorly from the acetabulum, and the femoral neck exhibits a forward rotation or anteversion as compared to its normal relation with the femoral shaft.

Atypical Cases. These cases, called "teratologic" by Le Damany[16] and "prenatal" by Steindler,[25] show a fully developed dislocation that had existed as such before birth. These are usually associated with other abnormalities, among which are arthrogryposis multiplex congenita, clubfoot, clubhand, spina bifida and others. This group, representing about 2 per cent of all cases of congenital dislocation of the hip, shows an equal distribution between the sexes. The femoral head may be located in any one of several sites about the acetabulum, usually anteriorly and superiorly. Korvin[15] felt that one third of these cases were related to poor fetal position or "spatial constriction" occurring during intrauterine life.

Others. There are cases in which some injury or pathologic state is present and may be etiologically related, such as obstetrical trauma, congenital syphilis, cerebral palsy or spina bifida with lumbar nerve involvement.

Anatomic Classification

The term "congenital dysplasia of the hip" includes preluxation, subluxation and dislocation of the hip. The authors consider these conditions to be stages in the development of the same condition. In any individual case the condition may be spontaneously arrested at either of the three stages, depending on the sum total of the endogenous and of the exogenous influences acting on the hip joint itself. Some of the

mechanical exogenous factors include positional forces applied to the hip, such as the Italian custom of binding the thighs and knees together with the hips in flexion and adduction, which favors dislocation. The Chinese custom of transporting the child with its thighs in the "frog-leg" position astride its mother's hip favors reduction of the femoral head into the acetabulum. Weight bearing on an unstable joint effects rapid progression to a more severe stage. Some of the endogenous factors include the degree of obliquity of the roof of the acetabulum; the integrity of the muscles acting upon the hip joint, especially the relative balance of power between hip abductor and adductor musculature; and the presence of an adduction contracture favors dislocation of the same hip.

Preluxation. This condition is the mildest or first stage in the development of congenital dislocation of the hip. It is asymptomatic. Physical findings are minimal and consist in slight limitation of abduction of the hip with or without asymmetry of the skin folds along the medial and posterior borders of the thigh. If asymmetry is present, there is an extra skin crease on the affected side. Roentgenographically, a slight distortion of "Shenton's line" (Fig. 96) may be seen. The significant finding is the presence of an abnormally great "acetabular index" (Fig. 95), indicating an increase in the obliquity of the roof of the acetabulum. When a unilateral frank dislocation of the hip is examined, the opposite "normal hip" is frequently the site of preluxation.

Subluxation. This condition represents an intermediate stage between preluxation and dislocation of the hip. In patients with bilateral involvement the physical findings are symmetrical; therefore the diagnosis without roentgenographic examination is difficult. X-ray findings are typical and are diagnostic. If the condition is recognized early and treated vigorously, good results can be anticipated.

Dislocation. Displacement of the femoral head from the acetabulum is the end stage of congenital dysplasia of the hip. Depending on the location of the femoral head, the dislocation may be either posterior (85 per cent or more) or anterior.[10, 18] The physical and roentgenographic findings depend on the amount of upward displacement, the location of the femoral head and the severity of associated adaptive changes.

PATHOLOGIC ANATOMY[8, 9, 10]

In the infant the cartilaginous rim of the acetabulum extends laterally to a width seldom appreciated because of its radiolucency. Ponseti[21] painted a strip of radiopaque material along the outer acetabular margins of a pelvis dissected from a stillborn infant with normal hips. Roentgenograms of this specimen (Fig. 92) emphasized the wide

lateral extent of the cartilaginous rim, particularly that portion contributed by the ilium which is destined to form the main weight-bearing segment.

The primary disorder which precedes the formation of subluxation and dislocation is the defective growth and development of the cartilage forming the superior portion of the acetabulum. The theories as to the origin of this defect are many, and a complete discussion thereof is beyond the scope of this paper.

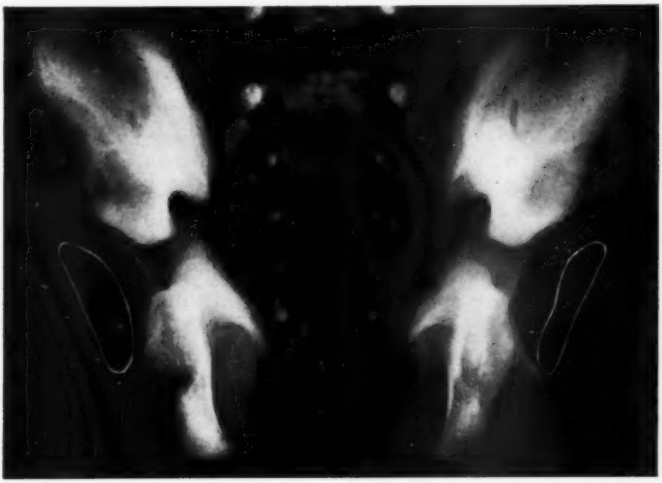

Fig. 92. Roentgenogram of the pelvis of a stillborn, full term infant with normal hips. The acetabular rims were painted with Diodrast to emphasize the wide lateral extent of the cartilaginous portions, not seen by usual technique. (Reprinted from Ignacio V. Ponseti: Congenital Dislocation of the Hip in the Infant. American Academy of Orthopaedic Surgeons Instructional Course, Volume X. Ann Arbor, Michigan, J. W. Edwards, 1953 [with permission]).

Most authors concede that the superior rim of the acetabulum is deflected upwards as the femoral head moves superiorly, and that the anterior and posterior portions of the circular cartilage are folded in simultaneously in much the same way as a circular rubber washer becomes an ellipse as pressure is made at diametrically opposed points. Since the cartilage cup is firmly attached to bone, and because pressure is applied at one relatively small area, a triangular-shaped acetabulum is formed, its apex pointing superiorly. The capsular attachments and ligamentum teres are slowly stretched without rupture, and the acetabular fossa becomes filled with fibrous and fatty tissue. The anterior and posterior portions of the acetabular rim more closely approximate one another, and the superior margin forms a grooved path for the move-

ment of the femoral head proximally and distally over its surface. The ligamentum teres hypertrophies, at times being more than 1 cm. in diameter, and so nearly fills the narrow hourglass constriction formed by the infolding edges of the glenoid labrum that the femoral head cannot gain re-entrance into its original acetabular fossa. After the femoral head has rested against the lateral side of the wing of the ilium a shallow depression may form. This is the so-called false acetabulum, and most often it assumes an oval shape in which the femoral head may slide up and down with weight bearing.

In the normal lower extremity of a young infant the femoral neck forms an obtuse angle (160 degrees) with the longitudinal axis of the femoral shaft. With normal growth this angle becomes less obtuse by about 25 degrees. The neck also points more anteriorly in the younger child than it does in the older. In the dislocated hip these normal changes do not occur, and the so-called reptilian hip with a femoral neck almost parallel to the shaft of the femur and with marked "anteversion" persists. These findings are roughly proportional to the degree of subluxation or dislocation present, being worse in the latter.[3]

In the neglected case a flat facet usually forms on the femoral head at the point where it contacts the false acetabulum in which it slides. The infolded cartilaginous edges become calcified and somewhat flattened, barring any possible re-entry of the head into the acetabulum. The muscles, fascia and capsular structures containing the unstable femoral head form a thick, fibrous retaining envelope which restrains the upward thrust of the femur as weight is borne on the limb. If frank dislocation of the femoral head has not occurred, various degrees of distortion of the joint surfaces may occur. In later life a hip which previously had shown no more than an oblique acetabular roof usually develops osteoarthritic changes in the femoral head and in the acetabulum. These changes are maximal at the point of greatest weight bearing and are irreversible.[2, 5, 17]

DIAGNOSIS

It is difficult to emphasize too strongly the importance of early diagnosis in these cases. The concept that a child must be over one or two years of age before treatment is started has proved false, the more current thought being that the earlier treatment can be instituted, the more easily and completely the soft cartilaginous, ligamentous and bony structures will grow to produce a normal hip joint.

History

Since dysplasia of the hip is not associated with pain, the only significant information obtainable from the family is usually that they

have noted a decrease in voluntary movement of the affected extremity. The family may or may not have noted an asymmetry in skin folds on the medial and posterior aspects of the thighs. Rarely the parents note a popping or snapping sensation in the hip region as the diapers are changed.

Physical Findings in Clinically "Typical" Cases

Preluxation. In preluxation there is frequently a decrease in voluntary movement of the affected extremity. If there is some slight upward dis-

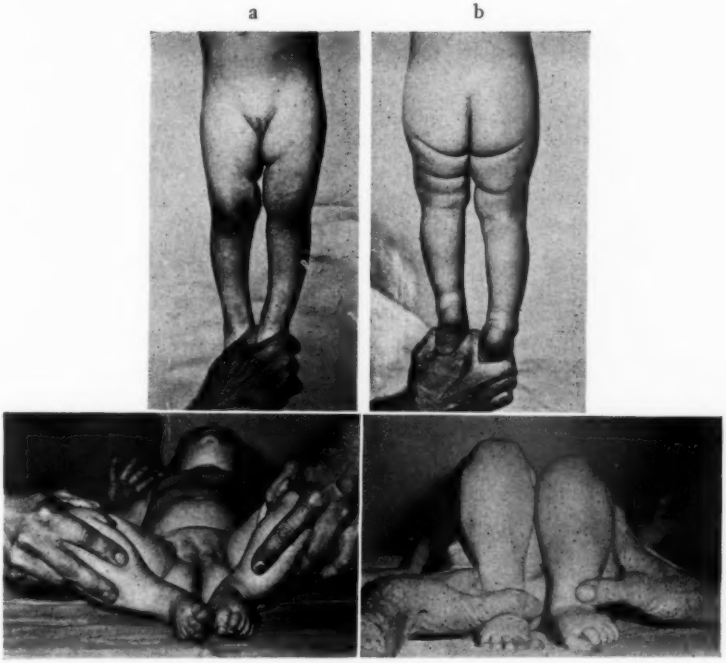

Fig. 93. Congenital dislocation of the left hip. *a, b,* Note asymmetry of skin folds of thigh, shortening of left lower extremity, and prominence of left greater trochanter. *c,* Limitation of abduction in left hip, asymmetry of skin folds with deepening of left gluteal cleft, and broadening of left perineum. *d,* Gross shortening of left femur is apparent despite the unintentional deviation of the thighs to the right.

placement of the femoral head, there will be shortening of the affected lower extremity noted on close inspection, and more rarely by direct measurement. Because of the skeletal shortening on the thigh there is a redundancy of skin and subcutaneous tissue, and this is manifested by

an increase in the number or depth, or both, of the medial and posterior skin folds or "wrinkles." There may or may not be limitation of passive abduction of the hip.

Subluxation. The foregoing findings are present to a more marked degree in subluxation. Shortening of the concerned extremity is frequently but not always noted. When present, it can be demonstrated by either of two techniques. The distance between the anterior superior iliac spine and the distal tip of the medial malleolus is less by direct measurement than on the normal side. A simpler and probably more effective method of measurement is illustrated in Figure 93, *d*, in which the child lies supine on a firm surface with hips flexed to 90 degrees and knees in full flexion. The examiner observes the heights of the two knees with the pelvis level and the knees in a vertical position. Differences in knee-hip measurements of 1 cm. are readily apparent with this technique.

There is more obvious asymmetry of the thighs in unilateral cases, and when the perineum is inspected, it appears to be broader on the side of subluxation. Limitation of abduction of either the extended or flexed hip is apparent when compared with the normal side (Fig. 93, *c*)

Dislocation. In congenital dislocation of the hip the resting position of the extremity is usually an attitude of mild flexion and adduction. Shortening of the affected extremity, as is true of the foregoing physical findings, is more definite, and more obvious to the examiner. The extra skin fold is usually well marked on the thigh in its middle portion (Fig. 93, *a*, *b*). Limitation of passive abduction is definite, as is broadening of the perineum on the affected side. In addition to these findings, palpation of the area of the hip reveals an absence of the usual firm resistance offered by the femoral head immediately lateral and distal to the point where the femoral artery crosses the inguinal ligament. However, in cases of anterior dislocation the femoral head is palpable immediately distal to the anterior superior iliac spine. In cases in which there is the usual posterior dislocation the femoral head may occasionally be palpable posterior and superior to its normal location.

The "sign of the jerk" is demonstrated when, while the hip is being passively abducted in a position of slight flexion, a palpable click or jerk is noted as the head of the femur suddenly slips over the acetabular limbus. This sign is diagnostic of dislocation. In the so-called prenatal or teratologic varieties of the "atypical" clinical type of dislocation and in the older "typical" cases, this sign is usually not obtainable because of the severity of capsular and periarticular contractures.

Telescoping is another sign of diagnostic import (Fig. 94). With the child supine on a firm surface, its hips and knees flexed, the examiner fixes its pelvis with one hand and alternately exerts traction and compression on the hip in the axis of the femur. If dislocation is present,

the femoral head glides proximally with axial compression and distally with traction in the manner of a telescope. This sign is diagnostic of congenital dislocation of the hip and is present in practically all instances of frank dislocation.

Nélaton's line is a straight line projected onto the lateral aspect of the hip, beginning at the anterior superior iliac spine and ending at the ischial tuberosity. The superior tip of the greater trochanter should be level with or distal to Nélaton's line. When it is proximal to the line, roentgenographic examination of the hip is indicated to rule out dislocation of the hip among other pathologic states.

Fig. 94. The technique of demonstration of telescoping of the left hip (see text).

The gait of a patient with dislocation of the hip is fairly typical. When the patient's weight is borne on the affected extremity, the femoral head glides proximally, thus providing an unstable fulcrum. In addition, the effective power of the abductor stabilizing musculature is lost by virtue of the unstable fulcrum as well as by approximation of the origin and the insertion of these muscles. As a result, the stabilizing muscles are unable to fix the pelvis solidly, and the opposite side of the pelvis tilts downward as the normal foot is lifted from the floor. This is the "Trendelenburg sign." To avoid the downward tilting of the pelvis as the normal foot is lifted in walking, the patient sways his superincumbent body laterally over the affected femur. Posterior dislocation of the hip, especially when bilateral, is characterized by an increased lumbar lordosis while standing or walking. This is caused by the flexion of the hip which results from the posterior and superior displacement of the point of weight transmission from the pelvis to the femur. To compensate for such hip flexion the lumbar spine must be extended when the patient assumes the erect position. On the contrary, in cases of anterior dislocation of the hip the point of weight transmission is an-

terior and superior to its usual site and an attitude of extension of the hip results. To compensate for this, the patient must flex the lumbar spine or flatten it when he assumes the erect position.[18]

Roentgenographic Findings

Roentgenographic examination of the pelvis, including both hips, is necessary to establish the presence and the degree of dysplasia of the hip. Care must be taken to avoid rotation of the pelvis and its concomitant distortion. On the anteroposterior projection the symphysis

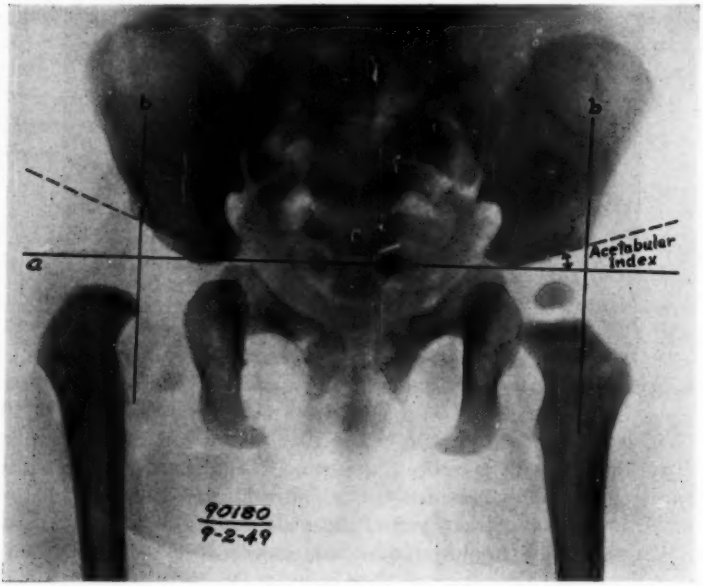

Fig. 95. Demonstrates (*a*) the horizontal reference line (Hilgenreiner), (*b*) the vertical reference line (Perkins), and the acetabular index (see text).

pubis should overlie the midline of the sacrum and the obturator foramina should be symmetrical in size and shape. Examination of the hips in the lateral or "frog-leg" position is of value in cases of subluxation and of dislocation. Special views are necessary to determine the degree of anteversion of the neck of the femur.

Arthrography using 5 to 10 cc. of Diodrast is a valuable adjunct. With this technique the true depth and contour of the acetabulum can be determined. Without opaque media in the hip only the ossified portion can be visualized. If there is a definite obstruction to reduction, such as an hourglass constriction of the capsule, this will be demon-

strated by the arthrogram. For a detailed discussion of the technique and interpretation of arthrography the reader is referred to the recent monograph by Hass.[10]

The most commonly noted roentgenographic changes in dysplasia of the hip are an increased obliquity of the roof of the acetabulum, and a retardation in the rate of ossification of the femoral head. The shadow for the latter appears at a later age, and the nucleus of ossification remains smaller than on the normal side until normal mechanics have been established in the joint.

Many roentgenographic signs have been described for use in diagnosis, treatment, prognosis and evaluation of results of treatment of dysplasia of the hip. Those described here have been found most useful in the authors' experience. In the hope of elimination of confusion the remainder are omitted from this discussion.

1. The *horizontal reference line*[12] (see Fig. 95) is a transverse line drawn through the triradiate or Y cartilages of the acetabulum and projected laterally on each side. In practice this line passes through the lowest extent of the shadow of each iliac bone. Normally, the femoral head lies well below the level of this line, and each femoral metaphysis is equidistant from the line. In frank dislocation the capital femoral epiphysis, if it has calcified enough to be seen on the roentgenogram, is found to lie above the line, and the top of the upper femoral metaphysis is closer to the line than that of the normal side.

2. The *vertical reference line*[20] (see Fig. 95) is a vertical line projected through the upper lateral edge of the acetabular rim, perpendicular to the horizontal reference line. A similar line is drawn on the opposite side of the pelvis. At least 60 per cent of the capital epiphysis lies medial to this vertical line on the normal hip. In subluxation less than 60 per cent of the femoral head is medial to this line, while in frank dislocation the entire epiphysis may be lateral to the vertical reference line.

3. The *arcuate reference line* (Shenton) (see Fig. 96) in its lateral portion is drawn along the inferior border of the neck of the femur. The medial portion of the line is drawn along the upper margin of the shadow of the obturator foramen projected by the inferior edge of the horizontal ramus of the pubis. These two lines appear as arcs of the same circle in the normal or preluxated hip, while on the subluxated or dislocated side they no longer appear as arcs of the same circle. This valuable reference line is distorted if the pelvis is not positioned carefully, or if the hip lies in external rotation.

4. The *acetabular index*[14] (see Fig. 95) is important because it indicates the depth of the ossified portion of the acetabulum and its obliquity; the latter is greater than normal in all stages of hip dysplasia. It is equally valuable in treatment as in diagnosis, for active treatment

should continue until the index is less than 30 degrees.[2] The horizontal reference line referred to earlier forms one limb of the angle. The other is formed by a line which begins at the distal tip of the iliac shadow and crosses through the most lateral margin of the acetabular shadow. The angle is read directly in degrees. It is felt that an acetabular index of 27 degrees at birth is the upper limit of normal. At one year of age, as pointed out by Hawkins,[11] the index is normally 22 degrees or less. Greater readings than those mentioned must be considered pathologic.

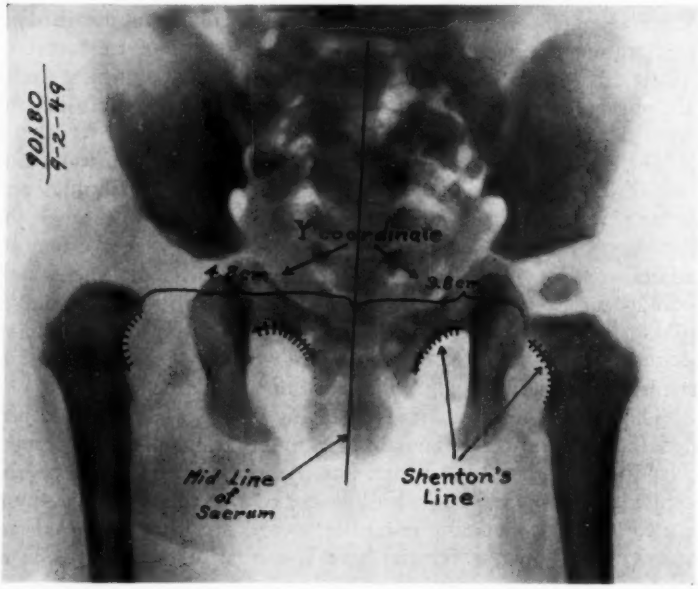

Fig. 96. Demonstrates the arcuate reference lines (Shenton) and the Y coordinate (Ponseti). (See text.)

In the experience of the authors an index greater than 30 degrees is associated with instability and resubluxation or redislocation, while an index less than 30 degrees is usually associated with stable reduction. The acetabular index may be within normal limits in the "atypical" cases.

5. The Y *coordinate*[21] (see Fig. 96) is read directly on a metric ruler. It is the distance between the midline of the sacrum and the most proximal and medial projection of the femur. It is an indication of the amount of lateral displacement of the femoral head. In the authors' experience it is mainly useful as a prognostic sign during treatment, and if the Y coordinate is greater by 0.5 cm. or more on the pathologic side, recurrence after treatment is likely.

6. In the *CE angle*[26] two radii are projected from the center of the capital epiphysis, one being perpendicular to the horizontal reference line and the other passing through the upper outer edge of the acetabular shadow. Normally the angle is 26 degrees. In subluxation the angle is diminished, and in dislocation it is reversed.

TREATMENT

The specific plan of treatment varies in the individual patients. The factors important in selecting the proper sequence of corrective measures are (1) the age at which the diagnosis is made; (2) the degree of dysplasia and/or type of dislocation present; (3) amount of progression prior to diagnosis.[24]

A preambulatory child with "typical" dysplasia without frank dislocation can be rather simply managed by the Frejka pillow splint. This maintains the hips in wide abduction, and with growth serial roentgenograms will reveal a progressive decrease in the acetabular index. These should be taken once every six to eight weeks until the index is within normal limits.

If frank dislocation is present, every attempt should be made to obtain reduction prior to the walking age (Fig. 97). This requires gentle manipulation, usually under anesthesia, and maintenance of the reduction by application of a plaster cast extending from the lower chest to the toes with the hip immobilized in the position of maximum stability, usually extension, abduction and internal rotation. A tenotomy of the shortened adductor muscles may be necessary preliminary to reduction, unless "guided abduction" as described by Craig[4] is used to stretch these muscles prior to manipulation.

The plaster cast maintains the thigh in the abducted position in which the joint is most stable, two or three changes being required during the six months period. After this some type of abduction brace, preferably that type which exerts its effect on the thigh itself, is used until the acetabular index returns to normal. If there is excessive anteversion of the femoral neck, it is usually corrected by subtrochanteric osteotomy four to six months after reduction.

Preliminary traction is usually necessary before reduction can be obtained in the child who has been allowed to walk on a dislocated hip. Skeletal traction is more efficient and is continued until the femoral head has been returned to a point opposite the acetabulum. Manipulation and reduction under anesthesia is then performed, and an adductor tenotomy is nearly always necessary in this age group. If excessive anteversion is present, a derotation osteotomy of the femoral shaft at subtrochanteric or supracondylar level should be done four to six months after reduction.

a b

Fig. 97. *a*, (*Upper*), Congenital dislocation of the right hip in a 17 month old female child. (*Below*), Immediate result following closed manipulation and reduction. *b*, Follow-up roentgenograms made at 5½, 8½, 14 and 19 months after reduction. Immobilized in casts 7 months and abduction thigh brace for additional 14 months.

In the child between three and six years of age extensive adaptive changes have occurred not only in the femur and acetabulum, but also in the soft tissues surrounding the articulation. Preliminary adductor tenotomy and skeletal traction are usually necessary. After the femoral head has been pulled down gradually to the level of the acetabulum and maintained at this level for seven to 10 days, reduction is indicated. In this age group closed reduction is frequently unsuccessful because of the fatty and fibrous tissue within the acetabulum, the hypertrophy of the capsule and of the ligamentum teres. As a rule, it is necessary to incise the capsule, débride the acetabulum, and excise the ligamentum teres before the acetabulum will retain the femoral head securely. If the acetabulum is inadequate to retain the femoral head, it should be deepened by means of a shelf operation, preferably a greenstick or incomplete osteotomy of the ilium immediately above the acetabulum throughout the proximal half of its circumference. The rim is then levered distally and maintained in the corrected position by properly placed wedges of bone. If there is excessive anteversion or valgus of the femoral neck, or both, they can be corrected by intertrochanteric osteotomy, either at the time of reduction or, as is usually done, subsequently before immobilization has been discontinued.

Patients over six years of age at the time of original treatment are doomed to some permanent disability of the hip. Several operative procedures have been devised to improve the stability of the joint, to improve the limp, and to diminish the pain which usually appears late in this condition, but the opportunity of restoration to normal appearance and function has been lost, and degenerative changes can be expected ultimately.

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FRACTURES OF THE FOREARM IN CHILDREN

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Fractures in children are as different from those in adults as are their metabolic and psychic problems. Epiphysial lines, rarefaction produced by blood vessels, dense growth lines, congenital fractures and pseudofractures, unique pathologic fractures and a host of other phenomena appear on the roentgenograms to complicate the diagnosis.

Except for transportation and farm machinery accidents, the causes for bone injuries in children are usually simple. The bone changes are characteristic, and the outcome is predictable. The principles of treatment are correspondingly simple. The fair healing of most fractures in children with indifferent treatment has led the unwary to neglect the fact that other fractures terminate disastrously unless expertly handled.

MECHANISM

At the distal end forearm fractures are commonly produced by indirect force, from a fall on the hyperextended outstretched hand. The force is the same as that which produces a Colles' fracture in an adult. In a child the roentgenographic appearance varies with the age. In young children there is usually a fracture of both bones, frequently greenstick, 1 cm. above the wrist (Fig. 98). From six to 12 a fracture of both bones is likely to be a little higher, with displacement of the distal fragment dorsally (Fig. 99). At this age the fracture may involve the distal epiphyses with partial or complete displacement.

Fractures of the middle third may be produced by direct or indirect

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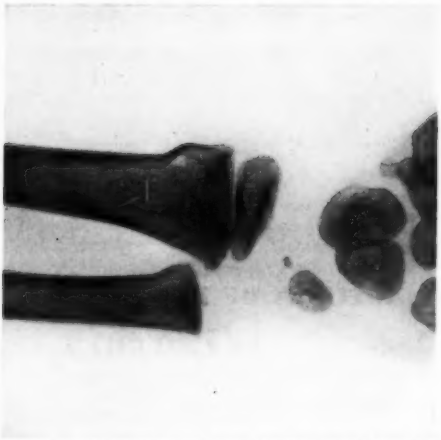


Fig. 98. Greenstick (torus) fracture of both bones just above the wrist in a boy 6 years old. Immobilization with a short cast or a splint for 2 to 3 weeks relieves the symptoms. Complications do not occur.

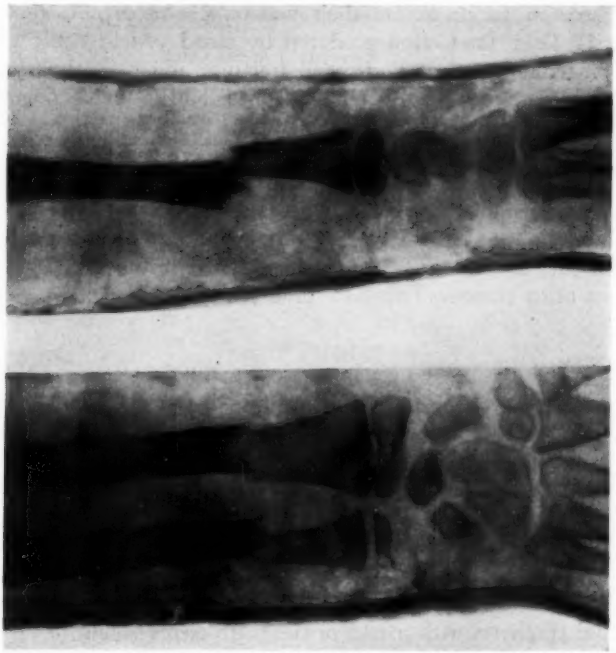


Fig. 99. Fracture of both bones in a boy 11 years old, characteristic of the age, 3 to 4 cm. above the wrist joint. Closed reduction failed. The fragments were immobilized in normal alignment with bayonet apposition and dorsal displacement of the distal fragments. Healing was prompt without deformity. A follow-up study 5 years later showed normal motion, strength and appearance. There was no radiologic evidence of fracture.

trauma. A direct injury may fracture the radius and not the ulna, or the ulna alone. Fracture of one or both bones is likely to be "greenstick" in children and angulated with the apex volar, producing a characteristic deformity (Fig. 100). The mechanics of the proximal third fractures are complex.

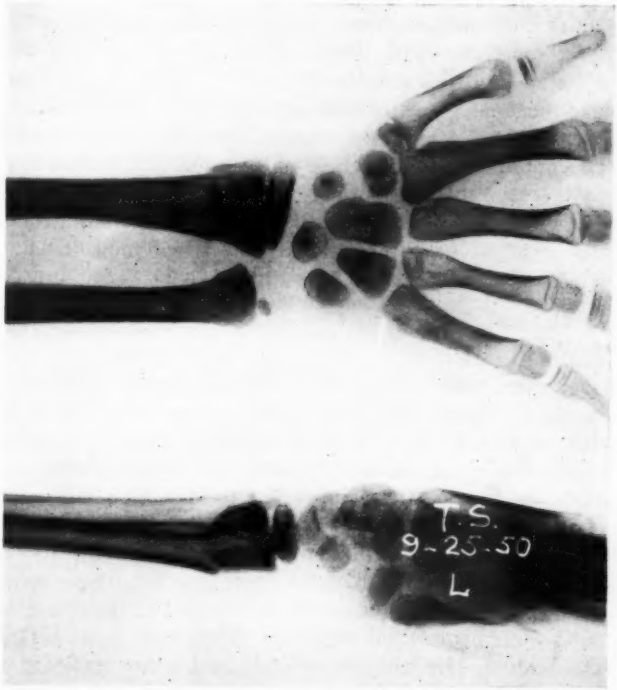


Fig. 100. Greenstick fracture, distal end, both bones of the forearm in a girl 7 years old. A cast was applied. The patient did not return for 5 weeks.

Refracture of both bones of the forearm at the same level is more frequent than generally realized. It usually occurs within the first six months after the original injury, but may be delayed longer. The reason is probably related not to any weakness of the bones, but to the child's habit pattern. Tumbles at a certain age are due to temporary awkwardness or carelessness. When the fall occurs, it is characteristically broken by the outstretched hand that is not carrying a toy. The same trauma is likely to be repeated. After a year or more, other activities cause other injuries.

DIAGNOSIS

The diagnosis is complicated in infants by the lack of an accurate history and by the lack of cooperation during the examination. Even the roentgenogram is difficult to interpret, because the ends of the long bones are composed largely of cartilage. The osseous centers appear at predictable ages. In older children the epiphysial plates introduce translucent lines which are sometimes difficult to distinguish from fractures. Fractures through the epiphysial plates are difficult to recognize if there has been a spontaneous reduction. Most important in this connection is to take roentgenograms of both arms in similar positions and each, of course, in two projections. Compare the roentgenograms of the injured extremity carefully with those of the uninjured. Failure to observe this rule is responsible for most of the errors in diagnosis of children's fractures. The torus fracture of the distal end of the radius may show only a wrinkle in the cortex, visible only when projected in profile (Fig. 98).

PROGNOSIS

General rules may be formulated for appraising a deformity and establishing a prognosis after the fracture of a growing bone. The degree of spontaneous correctability of angular deformities in long bone fractures of children is dependent upon three local variables: (*a*) the distance of the fracture from the end of the bone; (*b*) the age of the child; (*c*) the amount of angulation. The younger the child and the nearer the fracture is to the middle of the bone, the more accurate the reduction must be.

Apposition (the amount of end-to-end contact or engagement of the ends of the fragments) and moderate shortening are of little significance in children. The stimulus following a fracture results in accelerated longitudinal growth of the bone involved and sometimes of another distal to it. The result is similar to that observed in bones with chronic osteomyelitis. The mechanism is not entirely clear, and the results are not accurately predictable. There is no beneficent force which brings about a "compensatory" shortening.

A knowledge of prognosis, which is valuable in adults, is of the utmost importance in children who are going to continue to grow. It is this growth factor which makes fractures in children so different from those in adults. Further growth is usually a help in the correction of the deformities of bones that have united in a crooked position or with shortening. All too frequently growth is an inexorable force which produces deformity when the epiphysial cartilage has been damaged. These favorable and disastrous changes are predictable and should be understood by anyone who treats fractures in children.

TREATMENT

Principles

The principles of treatment are simple. Alignment is the chief requirement. The fracture should not be grossly angulated or rotated. Rotational deformities are inexcusable and should be corrected at an early stage of treatment. Slight angulation is frequently compatible with a perfect end result. This means that many impacted fractures in young children can be immobilized without reduction. On the contrary, angulated greenstick fractures near the center of the forearm bones of older children must be completely broken through and accurately aligned if permanent disability is to be avoided.

In most cases excellent results are obtained by closed reduction and a cast. Open reductions are difficult to justify. For lack of knowledge of sound conservative principles or to suit the convenience of some surgeon, unnecessary operations may have been done for years without serious complication. But even one unnecessary tragedy in a lifetime is reason enough to abandon such vicious operations.

In general, epiphysial fractures are best treated by closed methods. Unless there has been damage to the growing cells of the epiphysial plate at the time of injury, slight overgrowth is the rule, retarded growth an exception. After two weeks considerable displacement is usually to be preferred to forceful closed reduction or an open reduction.

Methods

The treatment varies with the level of the fracture. Open operation is rarely if ever justified in any case unless the radial head is also dislocated. Greenstick fractures of the distal third may be left unreduced if the angulation is not great (Fig. 100). The younger the child and the nearer the fracture is to the end of the bone, the more angulation one may leave unreduced. One should not ordinarily accept more than 30 degrees even in an infant, and this would be excessive in a child of six (Fig. 101). With growth and molding even greater angulation will eventually disappear (Fig. 102). At six or seven a 30 degree angle will produce a crooked arm for several months. The parents should be informed that the forearm will be bent at first, but will be perfectly straight eventually.

Distal Third. The closed reduction of a displaced fracture of both bones of the distal third of the forearm may require considerable skill. After a delay of a few days it may be impossible. If two or three attempts at closed reduction fail, there may be muscle interposed. This is not an indication for open operation (Fig. 103). Fixed traction should be applied (Fig. 104). The result will be a functionally normal

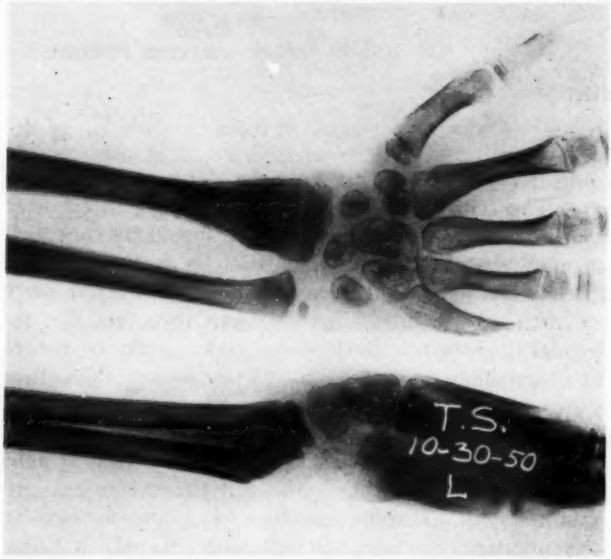


Fig. 101. Same patient as in Figure 100, but 5 weeks after the preceding roentgenogram, when the cast was removed. A checkup roentgenogram should have been taken in one week. The child had a bad cold. The angulation of 44 degrees remained. The bowing with the convexity volar was almost gone in 6 weeks.

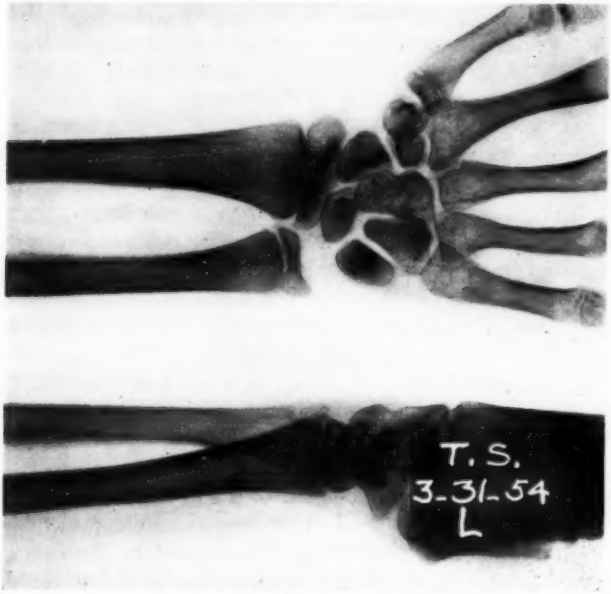


Fig. 102. Same patient as in Figures 100 and 101. Follow-up roentgenograms 3½ years after the fracture. The left forearm and wrist are clinically and radiologically normal. Moral: Angular deformities in the plane of motion of a joint of a young child straighten out to a remarkable degree.

forearm within six months (Figs. 105, 106) and normal bone architecture on roentgenographic examination within 18 months in a child of about six years (Fig. 107).

A fracture of the radius alone or of both bones at the distal end should be reduced in some degree of pronation and immobilized in a

Fig. 103. Characteristic forearm fracture in a boy 5 years old, sustained when his sled struck a parked car. After 2 unsuccessful attempts at closed reduction under anesthesia, alignment was maintained and overriding reduced to a minimum by traction. (Reproduced with permission of the Journal of the American Medical Association, 1952.)

cast extending above the flexed elbow (Fig. 108). The wrist should be in neutral flexion. Molding under the volar aspect of the radius to accentuate the normal curve is more effective than forced volar flexion of the wrist in maintaining reduction. Pronation will relax the pronator quadratus and, by twisting the radius "around the ulna," overcome the strong tendency to recurrence of the angulation with the apex volarward.

The cast should extend distally to the knuckles. In a child there is no need to permit free flexion of the metacarpophalangeal joints. Fingers do not become stiff after five weeks of partial immobilization. The hand portion of the cast will be battered to pieces with loss of position if one is too careful about freeing the child's hand.

Fig. 104. Banjo traction attached to a long arm cast with the elbow flexed. This was changed for a long arm cast without traction 17 days after the fracture.

After reduction and the application of a cast a roentgenogram that shows satisfactory alignment while the patient is asleep must be repeated in a few days to prove that angulation has not occurred from muscle pull. Another plate should be taken in two weeks. It is better to discover angulation in time to correct it easily than to find it when the cast is removed. Fragments that remain in malposition can be refractured at five weeks, but it is much easier at two.

Bayonet apposition in good alignment is not to be confused with angular deformity. The side-to-side contact produces no deformity or

Fig. 105. Same patient as in Figure 103. Except for slight swelling, the appearance of the wrist was normal on removal of the second cast. Clinically normal alignment is important in forearm fractures in children. (Reproduced with permission of the Journal of the American Medical Association, 1952.)

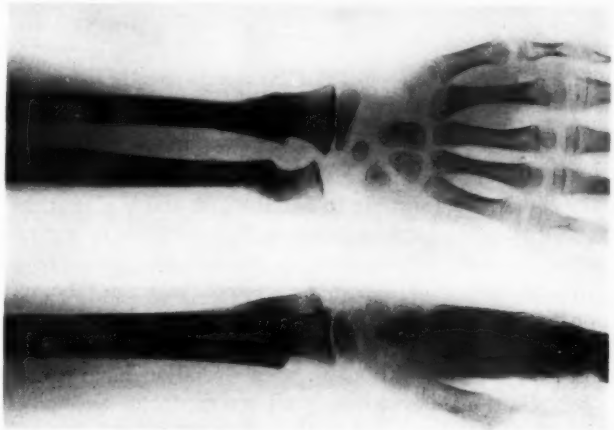


Fig. 106. Same patient as in Figure 103. Five weeks after the fracture there was good alignment, solid union and minimal shortening. (Reproduced with permission of the Journal of the American Medical Association, 1952.)

limitation of motion and is acceptable. Healing is solid in less time than with end-to-end apposition. Slight enlargement of the wrist rapidly disappears. Length should not be a problem. If there is appreciable overriding, it should be corrected and the position maintained with traction. When there is no tendency to displacement, there is no objection to midrotation. The wrist should be in neutral flexion (Fig. 108), slight palmar flexion rather than dorsiextension.

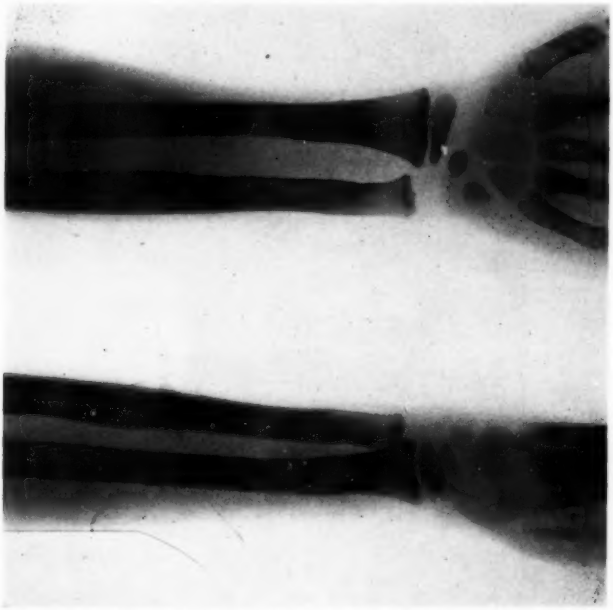


Fig. 107. Same patient as in Figure 103. Eight months after the fracture the roentgenogram shows almost complete restoration of the internal architecture. Clinically the forearm was normal. In the roentgenograms taken 18 months after the injury the left looked just like the right. (Reproduced with permission of the Journal of the American Medical Association, 1952.)

Occasionally it is permissible to cut the cast and wedge it, but care must be used to avoid harmful pressure. Pressure pads or pressure rods between the bones are not necessary in children and should be avoided because of the danger of ischemic necrosis. Casts must be split and spread, or bivalved if there is any threat to the circulation.

Many physicians who have taken the easy course and replaced the fragments in anatomic position by open operation have confessed to various complications. Nonunion is all too frequent. Several cases of pseudarthrosis at the distal end of the radius requiring bone graft have been reported by personal communications, and there are several

reports in the literature. Delayed union is common after operation. Deformity due to unequal growth is not uncommon (Fig. 109). Infection cannot be ignored. The scar is objectionable in a girl. Disastrous complications are rare, but are inexcusable when the results with closed methods are so good.

A reverse distal forearm fracture in which the force is applied with the wrist in volar flexion is somewhat more frequent in children than

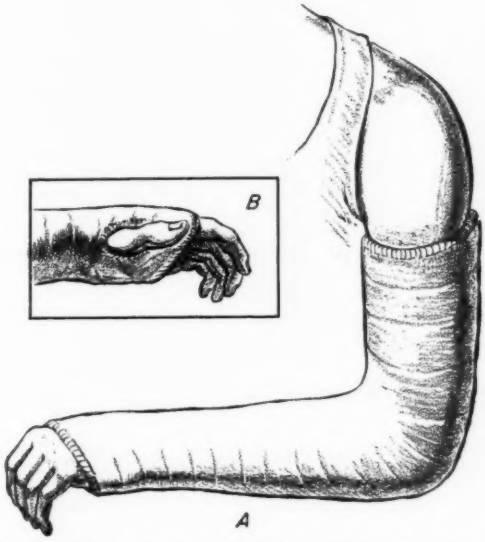


Fig. 108. Fractures of the distal third of the forearm should be immobilized in pronation; slight pronation for fractures of the radius only; and somewhere between mid- and full pronation for both bones. In a child the wrist need not be in the position of function. Extreme palmar flexion serves no useful purpose. Slight palmar flexion is the neutral position. The cast should be molded under the volar surface of the radius.

in adults, among whom it almost disappeared with the automobile crank. It occurs in children who fall on the back of the volar flexed hand. When the fracture is displaced, reduction is usually easy. This fracture should be put up in some degree of dorsiextension.

Middle Third. Fractures of the middle third of the forearm should not be allowed to remain angulated to any appreciable degree except in very young children. Reduction may be carried out as described for the distal third, usually with the forearm in midrotation. An angulation or rotation deformity here will persist for years (in older children, permanently) and limit pronation and/or supination. A true estimate of the amount of angulation frequently necessitates accurate roent-

Fig. 109, A.

Fig. 109, B. See facing page for legend.

C

Fig. 109. Complete closed fracture of the distal end of the radius in a girl 8 years old. *A*, Closed reduction attempted unsuccessfully on 2 occasions. Open reduction, one week after the fracture. She was discharged from the hospital in a plaster splint. *B*, In the plaster splint one month after the fracture. The position of the fragments is satisfactory. *C*, Eight years after the fracture. Note that open reduction has "done something" to the left forearm. (After closed reductions deformity does not occur.) There was bowing of both the radius and ulna, with the convexity laterally. Appreciable shortening of the forearm bones. Sixty degrees of limitation of pronation; 10 of supination. Other motions were normal. Slight loss of the carrying angle. Pain in the wrist with heavy work.

genograms of both forearms in symmetrical positions and several projections. The normal curves of the bones are deceiving.

Greenstick fractures at this level (Fig. 110) should be broken completely through by a quick, deft bending force over the surgeon's knee. Anesthesia is not required in a fresh case. After the fractures have been completed with an audible snap the fragments will remain in good position while the forearm is suspended by the fingers and a plaster

cast applied from the knuckles to midarm with the elbow at a right angle and the forearm in midrotation. There is no tendency to displacement. Cross union has not occurred in our series, and I have never heard of a verified case. If a greenstick fracture is not broken through, but merely bent to the straight position as advocated by some, the deformity will usually recur within the cast. Wedging of the cast or the use of a pressure pad to prevent the return of deformity is an invitation to Volkmann's ischemia. The appearance of pain is a danger signal here as at the elbow.

The position of a fracture of the middle third can be maintained by

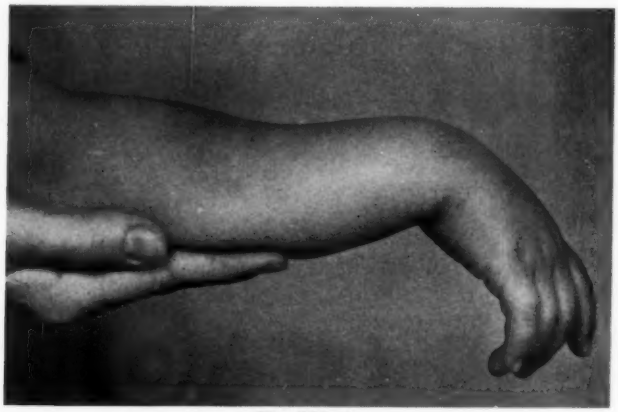


Fig. 110, A.

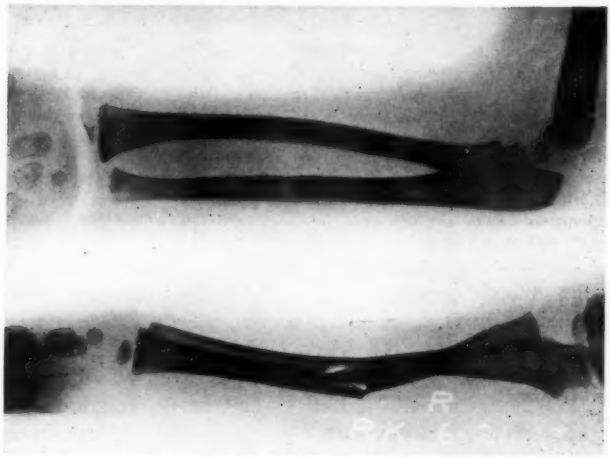


Fig. 110, B. See facing page for legend.

C

Fig. 110. A, Typical greenstick fracture of both bones of the forearm in a boy 3½ years old. The true angulation is masked by baby fat. B, Roentgenogram showing both bones broken through. A long cast was applied with the forearm in midrotation. The soft tissue hinge that Charnley describes is desirable, but a bony bridge will serve as a spring to reproduce the deformity unless it is broken through. C, Healed fracture 6 weeks later. Union is solid. The alignment is normal. The ulna normally bows with the convexity dorsally.

a simple cast with neutral version in most cases. Loss of alignment is no indication for open reduction. Satisfactory position can be restored with skin traction applied to the fingers with rubber bands (Fig. 104) over a banjo wire incorporated in the conventional arm cast with the elbow at a right angle and the forearm in midrotation. There is no risk of stiffness when the fingers of a child are immobilized with traction, but the danger associated with open reduction of this fracture cannot be ignored. Apposition is not necessary, and 5 mm. of overriding will be equalized by accelerated growth. Nonunion does not occur, even in the absence of treatment. Slight shortening is insignificant in the forearm.

Proximal Third. Displaced fractures of the proximal third are reduced with the forearm in complete supination (Fig. 111) and held in a plaster cast. Overriding is not objectionable, but alignment must be good. If there is a tendency to angulation, it must be corrected by traction applied to the fingers as suggested for fractures of the middle third. Bayonet apposition is acceptable, since there will be perfect

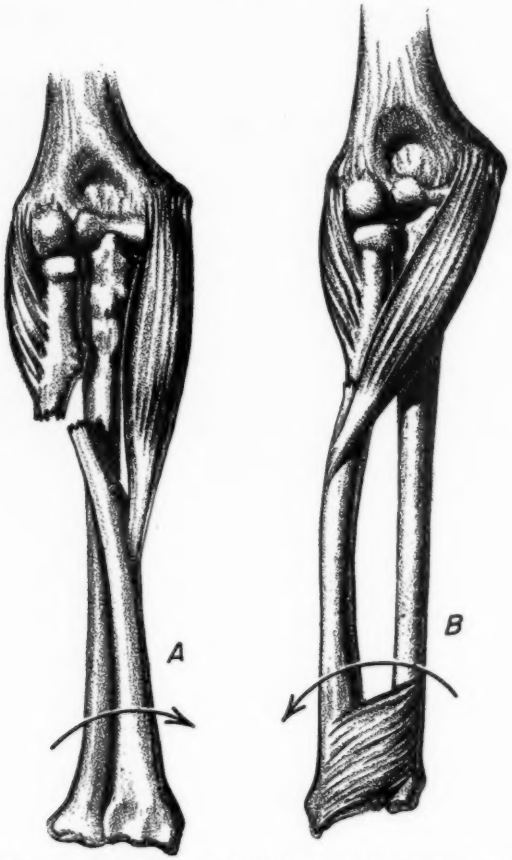


Fig. 111. Fractures of the radius or both bones in the proximal third should be reduced and immobilized in supination. A, The proximal fragment of the radius is controlled by the supinator muscle. B, The distal fragment is influenced by two pronators which must be stretched out by pronating the forearm to align the distal fragment with the proximal.

restoration of the bony contours in a few months and roentgenograms will look normal within a year. Function will be normal. Malposition is an indication for more efficient conservative measures and not for open reduction.

Single Fractures. Fractures of a single bone in the forearm at any level offer an interesting problem. If the fragments are displaced appreciably, an attempt should be made at reduction. The sound bone may be used as a lever and considerable force exerted in traction on the broken one. Pronation is useful in maintaining the position of a frac-

ture of the distal end of the radius; supination, of the proximal end. Accurate reduction hastens the return of function, but is not essential. There will be solid union even if the bone ends are moderately displaced. Callus will bridge interposed muscle masses in children. Restricted pronation and supination will eventually return to normal unless there is gross displacement in a boy over 12 (a girl over 10). Only when there is irreducible angulation at the middle third in a child over this age is open reduction justified. Internal fixation is unnecessary.

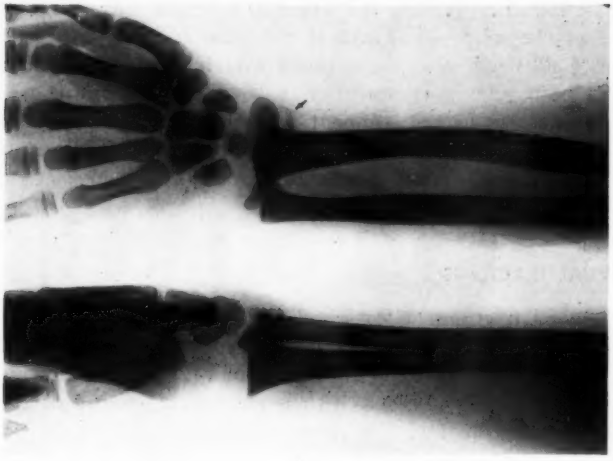
EPIPHYSIAL FRACTURES

Epiphysial fractures of the distal end of the forearm are unique in children and usually occur between the ages of six and 10. The radius alone or both bones may be involved. Sometimes displacement of the radial epiphysis is combined with a greenstick fracture of the ulna. Marked displacement of the epiphysial fragment is usually produced by direct trauma (Fig. 112, *A*).

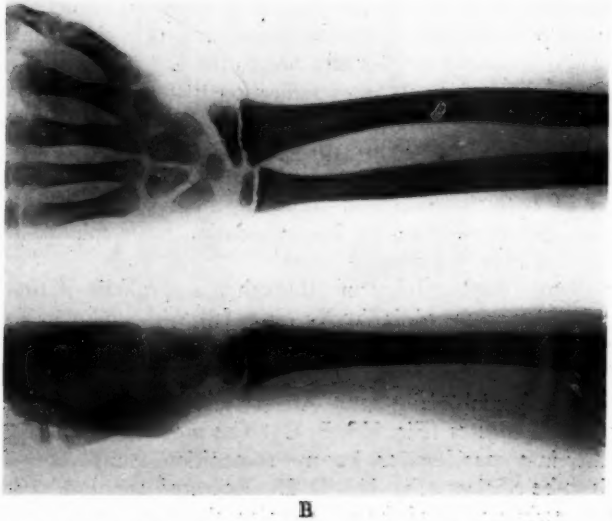
The *diagnosis* of a displaced epiphysial fracture is readily made by the roentgenogram. Occasionally there is spontaneous reduction. Then one should recognize the diagnostic importance of a tiny chip of metaphysis which usually remains attached to the dorsum or radial side of the epiphysial fragment. It is an indication that there had previously been displacement. When seen promptly, the fracture is easily reduced (Fig. 112, *B*). As in fractures through the distal end of the shaft, minor displacements need not be reduced. There will be prompt restoration of both function and form on the roentgenographic film. Slight overgrowth is common, but retardation from epiphysial injury is rare in this type of fracture.

The slightly displaced fracture through the epiphysis of the radius (Fig. 113, *A*) produced indirectly by a fall with considerable force on the outstretched hand may appear insignificant, but the associated crushing injury to the growing cells of the epiphysial plate is likely to cause retarded epiphysial growth. Angular deformity appears promptly as the bone elongates (Fig. 113, *B*, *C*). The prognosis should take this possibility into consideration. Prompt recognition is important if treatment is to be efficient (Fig. 113, *D*, *E*). Repeated osteotomies are preferable to early closure of the radial epiphysis to prevent the return of angular deformity. Not more than 5 mm. of elongation should be attempted at one operation. The radial deviation of the hand secondary to epiphysial fracture of the distal end of the radius must be differentiated from a true Madelung's deformity, which is usually bilateral, and asymptomatic until puberty.

Epiphysial fractures are treated like other fractures of the distal end



A



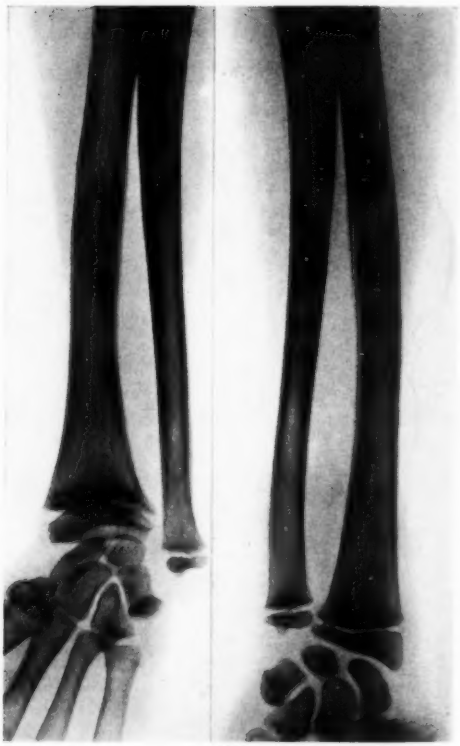
B

Fig. 112. A, Epiphysial fracture of the distal end of both bones of the right forearm in a boy 10 years old, caused by a fall from a straw stack. Note the chip of metaphysis attached to the radial epiphysial fragment (arrow). Reduction was easy under general anesthesia. A long cast was applied for 3 weeks. B, Roentgenogram of the fracture on removal of the cast. Examination 10 weeks later showed normal function. A 5 year follow-up showed normal function with 5 mm. of shortening of the right radius.

A

B

Fig. 113. See page 1118 for legend.



C

Fig. 113. See page 1118 for legend.

D

Fig. 113. See page 1118 for legend.

E

Fig. 113. A, Minimal displacement of the left distal radial epiphysis associated with a greenstick fracture of the ulna in a boy 7 years old was caused by a fall on the left hand. Quite properly, a cast was applied without further reduction. *B*, The injury must have been severe, with considerable damage to the growing cells of the left distal radial epiphysis, because this deformity appeared in the succeeding 5 years. *C*, Anteroposterior roentgenogram of both wrists, showing marked retardation of growth at the left distal radial epiphysis. *D*, One year after arrest of growth of the distal ulnar epiphysis and osteotomy with insertion of a bone block in the radius. The operation on the radius stimulated further growth at both ends of the bone. *E*, A good cosmetic result 7 years after the operation. Shortening of a forearm is not of great consequence. The deformity could not have been prevented, but could have been corrected earlier with less sacrifice of strength.

of the forearm. Open reduction is rarely necessary. The end result is influenced less by accuracy of reduction than by the amount of initial trauma to the growing cells of the epiphysis. Forcible or repeated reductions and particularly open reduction of an epiphysial fracture which is more than two weeks old should be avoided because of the danger of additional trauma to the epiphysial plate. After displacement of an epiphysis, realignment takes place subperiosteally, and the shaft soon follows the epiphysis to the new position. Apply the same rules for acceptability of the deformity as for fractures of both bones above the epiphysis.

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THE JUVENILE AMPUTEE

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Most child amputees fail to wear a prosthesis unless they receive specific attention and training. For reasons of function the failure to wear a prosthesis is much more common in the arm than in the leg. This failure to wear a prosthesis is due to the great adaptability of youth. The child compensates for the absent limb with comparative ease, and soon establishes a habit pattern which is difficult to break. This same great adaptability can be turned to the child amputee's advantage, since children are also more adaptable to the wearing of limbs when they are properly fitted and properly trained. Frantz and Aitken[1, 2] have characterized the child amputee as having three outstanding qualities: first, great adaptability; second, good brains; and third, poor judgment. From their experience the judgment must be supplied by the team of the physician, therapist and prosthetist, and through the parent who has received the proper instruction from this team. Because of their great adaptability, children who present severe or unusual stump problems can frequently be fitted under conditions in which adults would fail to wear the prosthesis.

REASONS WHY THE CHILD AMPUTEE PRESENTS A SERIOUS PROBLEM

The child amputee has received a great deal less attention than many other similar orthopedic disabilities in children. The progress that has occurred in the fields of cerebral palsy and poliomyelitis, for example, is not to be found in the field of child amputations. The apparent reason for this is that parents of child amputees invariably have a guilt complex about their child. If the amputation is congenital, they feel that they are somehow responsible, though they are not sure how. If the amputation has been traumatic, they feel that they have failed to prop-

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erly protect their child from danger. For this reason child amputees have rarely been gathered together in clinics where proper study of the problem could be carried out. A fortunate exception to this rule has been the work done by the Michigan Crippled Children's Commission at Grand Rapids under the guidance of Drs. Frantz and Aitken. The experience in Michigan shows that a carefully organized statewide program for the child amputee will result in a high percentage of successful limb wearers, and children who are much better fitted to take their place in society.

STUMP PROBLEMS ENCOUNTERED IN CHILDHOOD

In general, stumps which would be considered poor in adults and representing a difficult fit can be fitted with prostheses in children without complications. The difficulties encountered with scars, spurs, angulation of the stump, the presence of neuromas, or short stumps rarely lead to difficulty in children. This is due to the great adaptability of the child. Child amputees occasionally present the problem of overgrowth of a spike of bone or fibrocartilage at the end of the stump. Occasionally this spike will become sharp and pierce the skin. Only then is it necessary to remove the spike surgically. The use of an epiphysial arrest to prevent longitudinal growth of the bone is contraindicated. Epiphysial arrest will make the child's stump shorter and may interfere somewhat with circumferential growth of the epiphysis, and thus lead to an inadequate weight-bearing area, particularly in an amputation below the knee. Congenital amputees and children amputated under the age of 12 rarely present problems of phantom pain, and usually lack any type of phantom sensation.

COMPARISON OF ARM AND LEG AMPUTATIONS IN CHILDREN

The problems of arm and leg amputations are totally different. The functions of the upper and lower extremities are entirely different. In the human being arm function represents learned activity almost exclusively. It is significant in child development that the child has been walking long before he has learned any skilled activities in his upper extremity, and even though the basic arm activities may be possible to the child, the understanding of the activity may frequently be defective.

An example of this is presented by Gesell.[3] The child is presented with the problem of stacking up blocks to make four steps. Although the child is technically able to pile more than four blocks, one on top of the other, he fails to understand the concept of placing the blocks correctly to make four steps. He must mature considerably before he is able to construct the four steps.

In contrast to this, the function of the legs in walking on the level can be described as an almost instinctive behavior. This is particularly true if the amputation is below the knee. A child fitted with a proper leg prosthesis will walk almost as soon as a normal child. Because of these marked differences in upper and lower extremity function, they will be discussed separately.

UPPER EXTREMITY PROSTHESES FOR CHILDREN

At present it is felt that upper extremity amputees should be fitted about the age of four. With increased skill in training and with prostheses offering greater function it is felt that this age can be considerably reduced in the future. A few children have been successfully fitted at the age of one and one-half years. The problem of congenital arm amputation is particularly severe. Such children rapidly learn to make considerable use of the bare stump, particularly if it is a forearm stump. Congenital arm amputees should be conditioned to wearing some sort of device on their arm as soon as they begin to exhibit purposeful upper extremity activity. The device may initially consist of a soft leather cuff.

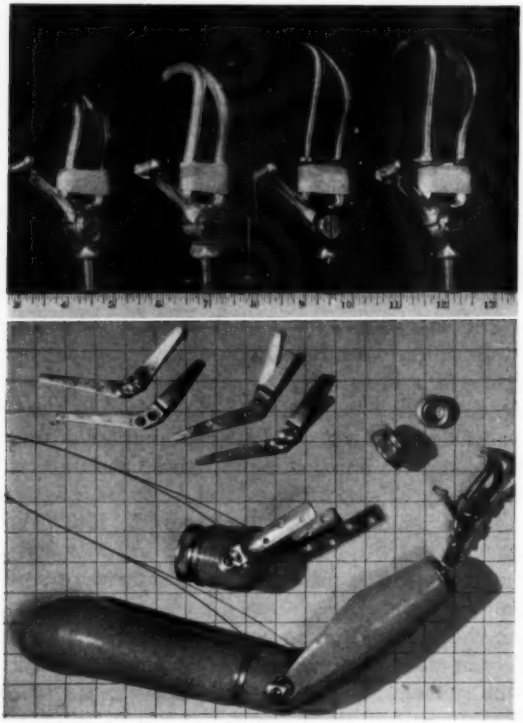
The two principles to be followed are, first, that the child should have a device which brings the amputated extremity out to normal length, and, second, that he should be encouraged to wear the device as much as possible. This will serve the dual purpose of preventing the formation of a habit pattern of using the stump without any prosthetic device, and, second, will accustom the child to wearing some sort of device which has some degree of usefulness to him. The child, of course, will not wear the device all the time, and it should be remembered that at this age it is difficult even to train the normal child to wear shoes all the time.

When the child first begins to wear the prosthetic device, a simple curved surface at the end which suffices for grasping objects with two hands may be suitable. When the child begins to play with toys, the attachment of a small "Alnico" permanent magnet at the end of the cuff will allow him to pick up toys which are stamped out of iron or steel. The toys can be released either by shaking them off or by pulling them free from the magnet. As the child's skill further develops, one of the small types of split hooks designed specifically for children should be attached to the prosthesis. As the child develops further motor skill, the terminal device should be attached to the usual type of shoulder harness.

Although most parents prefer to have some type of artificial hand because of its cosmetic value; it is felt advisable to use the split hook type of terminal device in most children since it presents considerable functional advantages, and the child will use the prosthesis only if it

offers him considerable function. All the modern artificial arms, including those available for children, are so designed that a hand or hook can be interchanged easily, depending on which type of terminal device offers the greatest advantage at any particular time of the day. The type of arm which will be prescribed depends upon the level of amputation,

a



b

Fig. 114. *a*, Four sizes of split hooks, suitable from child to adult size. *b*, Types of elbow and wrist devices for children. There is also a complete above-elbow type of arm. All are photographed against a 1-inch grid to indicate their small size. (Photographs courtesy of A. J. Hosmer Corporation, San Jose, California.)

and the uses which will be required of it. It is beyond the scope of this paper to present the complete picture on upper extremity amputations. The reader is referred to the *Manual of Upper Extremity Prosthetics*, published by the Prosthetic Devices Research Project at the University of California at Los Angeles.

The prosthesis for any amputee, child or adult, should be the result

of a conference with the surgeon, therapist and prosthetist, and should be devised in the form of a prescription with exact information on the type of terminal device, wrist disconnect unit, forearm component, elbow device, upper arm component, and harness type. The prescription should also include any necessary training for the amputee. A complete set of prosthetic equipment for children is available through any artificial limb shop. Children give prostheses rather rough treatment and a minimum of care. Most of the devices available for children are therefore of the heavy duty type.

The training of the child amputee is a field which needs a great deal of further work. Briefly stated, this training should be as complete as that of the adult arm amputee. The amputee is given first controls training, to teach him how to operate the various components of the artificial arm. When he has mastered his controls training, he is then put through use training. This consists of the various operations he will carry out in everyday life, such as picking up a glass or cup, the use of a knife, fork and spoon, and so on. The great advance in the future in child amputee training will come in the various types of devices used in the training and the sequence of training. It will be necessary to arouse and maintain the interest of the child in the use of his prosthesis at a much earlier age than is now possible.

THE JUVENILE LEG AMPUTEE

The child leg amputee presents a much simpler problem than the arm amputee. Leg function can be classed as almost instinctive. The problems in fitting the leg amputee are therefore similar to those in fitting the adult amputee. The training problem is more difficult, however, since the child has a greater tendency to develop bad habits, and is less conscious of the cosmetic features of gait. Children with amputations above the knee may encounter some difficulty with instability of the knee joint. When such a child amputee is first learning to walk, the difficulty with instability of the knee may be overcome either by aligning the knee joint in hyperextension so that it is quite stable during weight bearing, or by the application of a knee lock, which is unlocked only when he sits down. In either case the child rather rapidly learns to lock the knee by using the extensor muscles of the hip. Children with amputations below the knee walk well with the conventional type of prosthesis. Even in the case of the hip disarticulation, child amputees show remarkable skill in learning to walk with a proper prosthesis.

FOLLOW-UP CARE OF THE CHILD AMPUTEE

The success of any child amputee program depends to a great degree upon the follow-up care. The child will give his prosthesis heavy use at

a minimum of care and fall on it many more times than an adult. Because of this the follow-ups should be at periods not greater than three or four months. At the time of the follow-up examination the child should be examined by the team of the physician, therapist and limb fitter. The proper habits of use of the prosthesis, whether it be arm or leg, should be reviewed. Any bad habits should be corrected at each follow-up visit. The length of the prosthesis compared with the growing normal limb should be determined and corrections in length made as necessary. The prosthesis should be thoroughly inspected by the prosthetist. Any worn parts should be replaced and any repairs be made as necessary.

DISCUSSION WITH PARENTS

The parents of child amputees will require a considerable amount of sympathetic discussion and education. The guilt which most parents feel about their amputee child should be eradicated as soon as possible. This will probably take some discussion time over a number of visits. The parent must be guided through the fitting and training of the child. Many parents are repelled by the appearance of a prosthesis when they first see it, since they have usually imagined something which looks more like a normal arm or leg. It is only as they appreciate the functional value of the prosthesis that they become more reconciled to its appearance. It is important to impress upon the parents the necessity of their carrying out a continued training procedure, and a continuing correction of bad habits as the child may develop them in the use of his prosthesis. The parents must be encouraged to take an active part in the training, and be educated to do as much training as they are capable of.

It is important to stress in a discussion with the parents the future advantages to the child of wearing a prosthesis, since, particularly in young children, the advantages may not be apparent. They should also be encouraged to keep the child wearing the prosthesis as much of the time as possible, in view of the considerable advances which are sure to come in the next 20 years in the field of artificial limbs. The parents should further be urged to treat the child as normally as possible and to make a minimum number of concessions because of the amputation. Children with disabilities easily become badly spoiled if particular care is not taken in this field.

THE TEAM APPROACH

No one person has all the necessary skills to accomplish the complete care of the amputee. For this reason the team, consisting basically of the physician, therapist and prosthetist, is necessary for adequate care

of the child amputee. This is by no means the total team necessary. The pediatrician and psychiatrist may be added to the team as necessary. The social worker and other members of hospital personnel are also frequently involved. The advisability of the team approach to the amputee problem has been demonstrated by the Prosthetic and Orthopedic Clinic teams used by the Veterans Administration in the problems relating to both braces and prostheses.

Since 1945 an extensive program of research has been carried out by the Advisory Committee on Artificial Limbs of the National Research Council. Many improvements in the fitting of prostheses and the training of amputees have resulted from this research work. To make this knowledge available generally, a series of twelve schools in upper extremity prosthetics has been held at the University of California at Los Angeles under the auspices of the National Research Council Veterans Administration. One hundred and twenty prosthetists, 169 therapists and 182 physicians have been trained in these schools. Further research work is being carried out. Research in prostheses is continuing. There is increasing emphasis on the problems of the child amputee. It is anticipated that the next 10 years will show spectacular advances in this particular field of prosthetics.

CONCLUSIONS

1. Child amputees, whether congenital or acquired, offer a spectacular improvement in function when properly fitted and trained with a suitable modern prosthesis.

2. Prosthetic components are available for children. They can be fitted without the necessity of using any special type of equipment.

3. After fitting with the proper type of prosthesis the child should be given a period of training in the control of his prosthesis and the methods of using it in everyday activities.

4. The child amputee should be examined every three to four months to prevent the development of bad habits of prosthetic use, to keep the prosthesis at its correct length, and to keep the prosthesis in repair.

5. The team approach should be used in caring for the child amputee. The basic team consists of the physician, therapist and prosthetist, with additional members called in in consultation as necessary.

6. The parent of a child amputee should receive considerable attention, since the success of the program depends largely upon the proper parental cooperation.

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BONE AND JOINT TUBERCULOSIS IN CHILDHOOD

Recent Advances in Diagnosis and Treatment

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The discussion of any subject pertaining to newly developed medical or surgical treatment, in this era of rapid change, is fraught with considerable danger. When, added to this, the question of treatment of children is involved, the situation becomes even more complex. Furthermore, when one limits oneself to a subject which involves potential structural growth, one must become even more circumspect in one's attitude and statements. Therefore it is with apprehension that I discuss the present subject. The reader must be advised from the start that the basic treatment of old, and the well established basic methods of diagnosis of these lesions, still hold good and that the newer forms of diagnosis and treatment are in a state of flux. It is certain that tremendous advances have been made in the care of children suffering from tuberculous lesions of bone and joint. It is equally certain that further beneficial advances in the treatment of such lesions, as well as in their diagnosis, are but around the corner. Therefore this paper must be accepted factually as of the present, and not projected into the future years.

DIAGNOSIS

If one is to secure the optimum recovery in a child afflicted with a tuberculous bone or joint, all the past concepts of diagnosis must be revised. Indeed, the optimum result at present should strive for no less

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than complete normality. No longer should one await the night cries, sustained elevation of temperature, joint swelling, bony destruction and deformation upon which past diagnoses rested. No longer is it necessary to await the roentgenographic findings of joint narrowing, bone expansion, atrophy of contiguous bony structures, appearance of abscess shadows and bony caries (the precursor of deformation) commonly relied on in the past. Even the Mantoux test, negative or positive, is valueless in making a definite diagnosis in the early stages of this disease. Numerous patients have been seen with negative Mantoux tests who had definite joint tuberculosis. A positive test does not, with accuracy, designate the nature of a lesion involving a bone or joint structure.

This is not to say that the foregoing signs, findings and tests are entirely without value. They are of value, but of secondary import in establishing a diagnosis early. Probably the greatest factor in accuracy of diagnosis is a firmly implanted suspicion in a clinician's mind that lesions of tuberculosis in the structural system do exist far more frequently than is appreciated. The solitary bone or joint lesion in the child becomes suspect, particularly if the lesion fails to resolve under forms of treatment other than antituberculous and on therapy given with some other diagnosis in mind. There are, of course, many lesions of the osseous system which franky declare their nature as nontuberculous. Other lesions, lacking definite diagnosis, should be suspect as being due to tuberculosis. Many may actually be due to other conditions, but the suspicion of tuberculous involvement should be aroused. With the attention firmly focused on such a possibility in lesions with insidious onset and fairly chronic activity, definite instances of tuberculous involvement will be recognized at a date at which destruction of bone or joint structures has not progressed to such an extent that the pathologic state is not reversible.

Biopsy

Definite diagnosis of tuberculous involvement cannot be established without biopsy of one form or another. Excluding the spine, there are but few joints which are not readily exposed for surgical biopsy without damage. With all the clinical, laboratory and roentgen means at our disposal, relatively few cases will fall into the questionable group of which we speak. For these biopsy is not only justified but essential, and the earlier it is done the less will be the residual permanent damage.

The dangers of biopsy that existed in the past, no longer exist. With our newer antituberculous chemicals fear of entering tuberculous bone or joint foci no longer is necessary. Such incisions no longer disrupt as they did in the past, with devastating consequences. Biopsy demands

more than needle puncture in such lesions for accuracy. Tissue sections, modern culture techniques and guinea pig inoculations are essential. In most pathological laboratories the use of the guinea pig is gradually being discarded in favor of cultural methods. My personal feeling is that to secure the greatest percentage of accuracy, both culture and guinea pig inoculation are necessary.

Only in the spine is biopsy prohibitively costly as to possible sequelae. Fortunately, in this location, secondary soft tissue changes such as narrowing of the disk, due to sloughing, paravertebral thickening or alteration in the outline of density of contiguous vertebral bodies lead to as early and accurate diagnosis as is possible at this time. Other conditions producing these changes, such as brucellosis, are exceedingly rare and demand essentially the same surgical treatment and splinting of the spine by fusion that tuberculosis does. Furthermore, considerable accuracy of diagnosis as to brucellosis, coccidioidal infections, and so forth, can now be secured through laboratory findings.

Increasing suspicion and pointing to the necessity of definite and rapid diagnosis by biopsy is the racial and social background of the child. Even without proved contact with tuberculous patients, it must be assumed that such contact exists in our larger centers. Added further that such a child has a lesion of a solitary joint, chronic in nature, gradually progressive and lacking the laboratory and roentgen findings of other definite lesion, invasion by the tubercle bacillus becomes highly probable.

Therapeutic Tests

Finally, diagnosis by therapeutic test using the new antituberculous drugs can be a highly efficient way of establishing the etiology of a lesion. We would, however, warn against such therapeutic tests, for several reasons. After such arrest of a lesion absolute certainty of diagnosis is lacking. The stigma of tuberculosis will be placed upon the child, who may not have had tuberculosis. Isoniazid and iproniazid carry with them certain toxicities which should be avoided when possible. To date, we do not know the action of these materials in certain lesions other than tuberculosis, and such action may, in the future, prove disastrous. (Note cortisone and tuberculosis.) Without a definite diagnosis one is at a loss as to the length of time one must use the antituberculous chemicals in any individual case. Furthermore, one may have been mistaken in one's premise as to diagnosis and other lesion be present and progress to disastrous extent while the antituberculous chemicals are being given in vain.

One should accept the fact that diagnosis should be unequivocally made before the use of antituberculous chemicals or medication. In

most instances, if isoniazid or iproniazid is given, within a few days tissue sections and cultures may be of no avail. In early lesions the use of iproniazid for two or three days will prevent cultural determination of the tubercle bacillus even when present, and, in the child, synovial soft tissue or bony pathology becomes so rapidly altered that tissue section cannot give an unequivocal answer as to diagnosis. First, biopsy; then treat! How soon should one begin antituberculous medication after biopsy?—immediately; the same day. If tissue has been removed, it is only a matter of a few days, and an answer may be secured. In such a short period, delay can do no damage if other lesion exists, and the toxicities that may arise from antituberculous medication will be negligible.

TREATMENT

One of the benefits of advancing years is the ability to gaze back on personal experience. Only in this way can one tend to advance with safety. Well we remember the gradual improvement in the care of such patients, both as to their active disease and in the field of preventive medicine. Such matters pertain to treatment, even as of the present. Let down the bars but a little as to the testing and destruction of tuberculous cattle and the pasteurization of milk, raise but a little the economic barrier to good nutrition for our entire population, crowd but a little further the adults and children together, and once more tuberculosis will be rampant. Tuberculous lesions of bones and joints are slowly and steadily disappearing, but they are disappearing not because tuberculosis is nonexistent, but because foci of distribution are being stamped out. The greatest advance in treatment of such lesions still goes back many years to the preventive measures just mentioned. Make no mistake. We have advanced in the treatment of the individual lesion, but to no similar extent as was made in the prevention of the development of such lesions. Public health and preventive measures were, and are, the greatest advance in the control of tuberculosis in children and, with it, the control of associated bone and joint lesions.

Even with the tremendous decrease in incidence of bone and joint tuberculosis that followed the institution of adequate and universal control of milk and milk products, a really considerable pool of tuberculous lesions continued to exist in children. Gradually improving economic status of the people in this country caused a definite decrease in incidence of such lesions. Better nutrition, better housing, more widely spaced population, better education of the people as to disease in all its forms, and segregation of the active tuberculous patient have all exerted a favorable influence on the decline of this disease. With the decline in general incidence has gone less frequent contact of children

with the tubercle bacillus. Thus a decrease in tuberculosis in children and, of course, in incidence of bone and joint lesions occurred. As in so many matters, so in tuberculosis: the welfare of the people becomes the welfare of us all. This again is a direct factor in treament of tuberculosis.

Surgical Procedures

The first advance in the direct treatment of specific lesions of bone and joint tuberculosis in children was the recognition by Hibbs that internal splinting by arthrodesis of joints involved with tuberculosis markedly shortened the progress of the disease, greatly lowered the mortality, greatly reduced the rate of secondary complications and markedly reduced the amount of deformity generally resulting. Although ankylosing surgical procedures were a great advance in the treatment of such lesions, surgeons were still left with many insurmountable obstacles. Frequently, lesions were so acute that operation could not be performed, and surgical procedures had to be deferred until subsidence of the acute manifestations. During this period further destruction occurred, dissemination to other parts of the body took place, or death ensued. Control of the active lesion pending ankylosis was frequently impossible. Prevention of the breakdown of wounds due to tuberculosis, and secondary infection occurring through the line of incision, could not be controlled. Metastatic spread of the tubercular process due to the surgical trauma, while infrequent, occasionally did occur. Despite this, during this period, with conservative supportive therapy, plus surgical fixation, notable advance in the control of bone and joint lesions was achieved. A feeling of considerable frustration, however, existed.

Medical Treatment

Streptomycin. We shall not soon forget the tremendous advance in treatment that occurred with the introduction of streptomycin, discovered by Waksman. It seemed that we, who were especially interested in tuberculous lesions of bone and joint, lived in another world and could begin to breathe freely. Reduction in morbidity and mortality in the treatment of these patients was immediate and tremendous. Gradually, however, it became apparent that benefits secured by streptomycin had to be paid for in other ways. Gastroenteritis, exfoliative dermatitis and the development of permanent or partial deafness in as high as 20 per cent of the patients demanding prolonged treatment began to appear. Resistance of the tuberculous bacteria to streptomycin developed. This did not, to us working on bone and joint tuberculosis at Sea View, appear such a formidable obstacle. While with strepto-

mycin a great improvement occurred in the results of treatment of such patients, much was still desired.

And so we come, perhaps, to the part the reader wishes most. We have developed the treatment of tuberculosis slowly and gradually for a definite purpose. First things first. Modern antituberculous chemicals cannot be successfully used alone. Preventive medicine, general nutritional improvement, surgery when indicated, streptomycin occasionally to delay growth of the tubercle bacillus and to destroy secondary invaders, rest, quiet, time for healing of tissues badly damaged, all are as important now as in the past and are part of the modern treatment of tuberculosis of bones and joints.

Isoniazid and Iproniazid. In 1951, by chance, we at Sea View were favored with the opportunity of using isoniazid (Rimifon) and another chemical, iproniazid (Marsilid), on an experimental basis. Animal cultural experimentation had seemed to show them effective in the treatment of tuberculosis and with toxic manifestations that could be controlled. The children and the adults at Sea View (and later at St. Luke's) proved the worth of these two drugs. These materials were in the beginning in short supply, and iproniazid was known to be the more toxic. Fortunately for us, as orthopedic surgeons, iproniazid became available for us to use. I say "fortunately," since, later in a contrasting series of patients, it became evident that in bone and joint tuberculous lesions, in which dead fragments of osseous tissue frequently lie encased in necrotic material as foreign body irritants, iproniazid, while more toxic, was shown to be much more effective than isoniazid. Essentially on a clinical basis, iproniazid has been shown to be more than 20 times as effective in the control of orthopedic lesions than isoniazid. Gradually in the treatment of such lesions at Sea View and St. Luke's, we have almost ceased to give streptomycin or isoniazid and now rely on iproniazid as our primary therapeutic agent for tuberculous bone and joint lesions.

Iproniazid, when given in adequate doses to control the tubercle bacillus and heal bone and joint lesions infected therewith, produces a multiplicity of toxic symptoms in any considerable group of patients. These toxicities have been well described in other publications. They are all reversible. None are of great importance except that of psychosis. In the use of iproniazid, within dosage limits to be described, personality changes and psychosis will develop infrequently. On cessation of the medication all such patients have recovered. On any attempt to exceed the limits of medication advised, development of psychosis is fairly frequent, and may be severe, requiring even institutionalization for several weeks before recovery. Children as a rule have few complaints as to toxic symptoms. They probably do not notice and report occasional dryness of the mouth, hesitancy of urination, mild vertigo,

and so on. Personality changes usually develop slowly and must be watched for. The child may become somewhat excitable or morose or difficult to control in its association with other children. The actual development of psychosis in a child has been rarely seen by us. Perhaps this is because the child accepts hospital surroundings as the normal mode of life very readily, while adults do not. Psychoses developing in adults have been seen only during hospitalization. We have not had the experience of seeing psychosis develop in either an adult or a child while under the influence of normal home surroundings. Therefore, within reasonable dosage limits in children, iproniazid has proved an exceptionally safe form of antituberculous medication. With its use, deafness, gastrointestinal upset and skin eruption, occurring with streptomycin, are absent.

With the use of iproniazid in children, for the first time we have been able to save proved tuberculous joints with normal function. With streptomycin we were never able to approach this. Such lesions were frequently seen to recede on streptomycin, but on cessation of medication reactivation of disease usually occurred, if the patients were observed over a long enough time. With iproniazid, not every tuberculous joint in a child can be saved with good function. Often destruction has progressed to too great a limit to mechanically permit such salvage. Though we have had children treated for tuberculous bone or joint lesions under observation for almost four years, even this period of time is not great enough to disclose the compete truth as to permanency of recovery. All we can say is that we have numerous instances of children with proved tuberculous bone or joint lesions that have healed without surgery and have remained healed for one to three years after removal from iproniazid medication. There are also many patients in our series who were treated by surgical incision and expression of abscess material and by joint ankylosis. When spinal disease exists, we believe fusion to be always indicated.

In addition to the control of the tuberculous bacillus and its destruction, iproniazid produces many other clinical manifestations of benefit. Fever, when it exists, is reduced, appetite re-established, weight gain is remarkable, and pain when present is considerably relieved. These accessory beneficial effects in themselves would provide a favorable situation for recovery, did not the drugs actually have a direct effect on the tubercle bacillus.

In the treatment of the child with tuberculous bone or joint lesion accuracy of amount of medication is a prerequisite to success in therapy and avoidance of complications. It is surprising that views previously expressed, based on rather short, albeit intensive, evaluation of statistics and material, should not now be greatly altered.

In a child we would start medication at an accurate 4 mg. of iproni-

azid per kilogram of body weight per day given in three divided doses at eight hour intervals. We would, in most instances and unless the infection was severe, reduce this dosage within 48 to 72 hours to 3 mg. of iproniazid per kilogram of body weight per day in three divided doses. We would then continue with this same medication indefinitely unless personality changes developed or hypertonicity, indicated by markedly hypertonic reflexes or clonus, indicated further reduction. In such few cases the dosage may be reduced to 2 mg. per kilogram of body weight per day in three divided doses. We have seen no child whom we could not continue indefinitely on this lower dosage, and most of the children may be continued indefinitely at the rate of 3 mg. per kilogram of body weight per day. Medication will of course have to be increased as weight is gained.

SUMMARY

There is no modern treatment of bone and joint tuberculosis. Actually, through the years, a logical sequence of events has occurred, beginning with good supportive therapy and attempts to prevent deformation, enhanced by surgical procedures, broadened by streptomycin and now again further improved by the antituberculous chemicals, isoniazid and particularly iproniazid. Even to date, considering the improvement in therapy accompanying the use of iproniazid, further advance is possible. While morbidity and mortality in children from this dreadful affliction are at a minimum, a minimum still exists. Other agents to control this dread disease will be found until there is, as in the case of diphtheria, practically no minimum left.

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THE PROBLEM OF UNEQUAL LEG LENGTH

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The early recognition of discrepancy in length of the lower extremities in the growing child is particularly important since at this age appropriate management may prevent a disabling abnormality. A small discrepancy in the child may be the first evidence of a progressive disturbance in growth which may reach major proportions; on the other hand, a small discrepancy may be static and of little concern. It is important, therefore, not only to detect the differences in length, but also to determine their cause and to recognize their potential degree and clinical significance. Certain abnormalities require careful serial observation and subsequent modification of growth to correct the abnormalities. These must be recognized and followed accurately. In others such detailed observations are unnecessary.

CAUSES OF DISCREPANCY

Some discrepancy in leg length is measurable in 65 to 70 per cent of any normal group of human beings, provided the technique of measurement is accurate enough. In the measurement of 293 subjects from age two years to maturity by our group, it was found that 40 per cent had the right side shorter by greater than 0.1 cm. and 25 per cent had the left side shorter by this same amount. This difference is of neither statistical nor functional significance until it reaches a certain level. In adults and in children 12 years of age or over this borderline falls at

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about 1 cm. difference, but is less in the earlier years. For example, from birth up to two years a discrepancy of as little as 4 mm. may be significant.

Discrepancies of significant degree may arise from either overgrowth or undergrowth. On occasion it is difficult to determine which is the factor.

Overgrowth

Overgrowth is less common than inhibition of growth and is less likely to be of extreme degree. In many instances it seems to be asso-

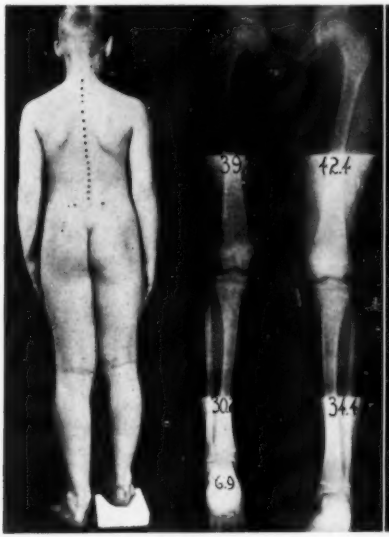


Fig. 115.

Fig. 116.

Fig. 115. Congenital hypertrophy in a boy aged 11 years. Left leg 2½ inches longer than the right, with a total difference in bone length of 6.9 cm. (2.7 cm. overgrowth in the left femur and 4.2 cm. in the left tibia). A longer and larger left leg was noted in this boy at birth, but no other abnormalities were ever demonstrated.

Fig. 116. Overgrowth of the right leg caused by neurofibromatosis in a 12½ year old girl. The tumor extensively involved the entire right lower extremity, which became not only larger in circumference, but also as much as 4¾ inches longer than the left.

ciated with increased circulation to the epiphysis for one reason or another, but in others the factor entering into the overgrowth is less evident. In some, such as a congenital hypertrophy, it seems to be associated with the differential growth capacity of the epiphyses and the extremities. This congenital hypertrophy may be a hemihypertrophy

in which the whole half of the body is of greater size than its fellow; it may involve one extremity, more likely the lower, or on occasion it involves a localized part of an extremity. If it involves a localized area, there may be marked distortion associated with the abnormality, but in most instances the hypertrophy is general and involves all factors

Fig. 117.

Fig. 118.

Fig. 117. Overgrowth after severe bone infection. This 11½ year old girl had acute osteomyelitis of the left femur with extensive destruction and a massive involucrum surrounding the entire shaft of the bone when she was 5½ years old. Lengthening of the affected femur occurred only during the activity of the disease, which lasted 11 months. Five years after the infection had healed (above) the involved femur was still 2.2 cm. longer than its mate, and a 1-inch raise was necessary to balance her back.

Fig. 118. Overgrowth of the right femur following fracture at the age of 9½ years. This 13 year old boy had a fracture of his right femur at the juncture of the upper and middle thirds. Excellent alignment of the fragments was produced with traction. Overgrowth occurred on the side of the fracture amounting to 2.4 cm. in the femur and 0.7 cm. in the tibia. No increase in discrepancy was measurable 17 months after injury.

in the extremity, which has quite normal appearance except for its size (Fig. 115). In such hemihypertrophy there is usually an increased increment of growth throughout the growing period, so that in planning the treatment of such a patient one must consider the potential discrepancy at maturity rather than the discrepancy at a particular age. In our cases, hemihypertrophy is more likely to occur in families in which there is a history of twinning. This may or may not be pertinent.

Increases in circulation to the epiphyses arising from many causes may give rise to overgrowth. Overgrowth occurs in some conditions in which the circulation factor is evident, as in arteriovenous aneurysm or communication. It arises in other conditions in which the circulatory factor is not so apparent. In chronic osteomyelitis, for example, in which the metaphysial area and the adjacent diaphysis are involved by chronic inflammation, the associated long-continued hyperemia in this area and in the adjacent epiphysis often results in overgrowth, provided the plate itself is not injured (Fig. 117). Occasionally such overgrowth is of a degree requiring surgical correction of the difference. In the same manner chronic inflammation of a joint may stimulate overgrowth in the adjacent epiphysis. Chronic monarticular arthritis of the knee, for example, may stimulate growth of the femur at the distal epiphysis and the upper tibial epiphysis as well. Such stimulation to an extent of ½ to ¾ inch overgrowth is not unusual in the growing child, provided the process goes on for a considerable time.

Fracture of the shaft of a long bone may produce overgrowth. Particularly does this occur in a fracture of the femur (Fig. 118). The overgrowth probably arises because of the interruption of the nutrient vessel and a compensatory increased vascularity in the metaphysial and epiphysial vessels. The reaction may not be quite as simple as this, since after such a fracture of the femur there may be some overgrowth in the tibia as well. Ordinarily this overgrowth is not of a disturbing degree, but on occasion it is enough to warrant secondary procedures to equalize the length. It is ironic, indeed, to have reduced a fracture of the femur perfectly only to have overgrowth of an amount to produce a distinct list in the spine or an excess growth warranting a surgical procedure.

Overgrowth occurs in various other conditions such as neurofibromatosis, in which the mechanism producing it may not be clear. In some, at least, it is associated with increased vascularity in the neurofibromatous elephantiasis of the extremity, and in at least one instance it was associated with a lack of control of these vessels by the autonomic nervous system (Fig. 116). In general, most of the instances of overgrowth other than those of congenital variety, in which apparently the growth potential of the part is abnormal, are associated with increased vascularity of the epiphysial area from one source or another.

Undergrowth

Undergrowth, or discrepancy in which inhibition or lack of growth is a factor, is seen more frequently than overgrowth. The most common cause of shortening of clinical significance is poliomyelitis. Congenital abnormalities, however, give rise to the most disabling forms of dis-

crepancy. Congenital absence of a bone or of a part of a bone may give rise to extreme discrepancy. A common entity is a congenital absence of the upper portion of the femur, in which all the upper portion is missing except the ossification center of the head (Fig. 119). Absence of the tibia or of a part of the tibia is not too unusual. Congenital absence of the fibula with a four-toed incomplete foot and a short tibia

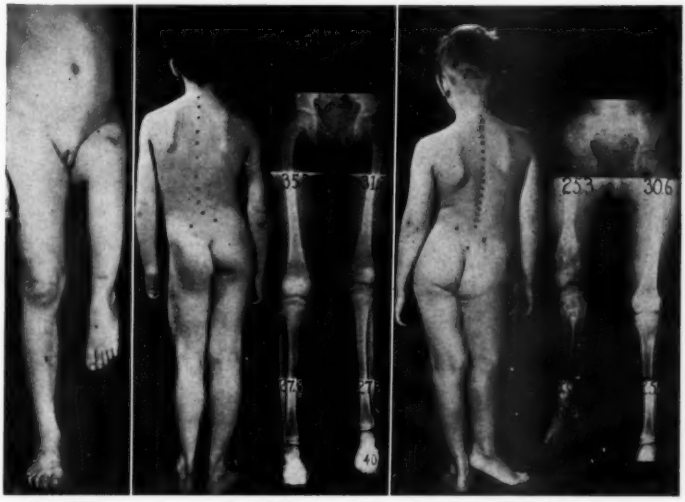


Fig. 119. Fig. 120. Fig. 121.

Fig. 119. Congenital absence of the upper two thirds of the left femur in a girl aged 7 years, with associated severe shortening (25 cm.) of the extremity and flexion contractures of the hip and knee.

Fig. 120. Congenital atrophy of the left femur in a boy in whom a constant, slow rate of inhibition was observed in the femur throughout his growing years. At the age of 8 years and 9 months (above) the right femur was already 3.5 cm. shorter than the left, and the tibia was 0.5 cm. shorter.

Fig. 121. Chondrodysplasia principally affecting the epiphyses at the right knee, causing severe growth inhibition in a boy aged 7 years. The right leg is already 10.3 cm. shorter than the left, with the discrepancy about equally divided between the femur and the tibia.

is a rather common abnormality. All these severe deformities present special clinical problems which require individual consideration.

Congenital shortening is more often of milder degree in which the growth potential is less on the affected side and the proportions are such that it is evident that the abnormality is of the shorter extremity rather than in the long one (Fig. 120). It may on occasion be difficult to determine whether the difficulty is due to overgrowth of one extremity or undergrowth of the other. A particular epiphysis may or may not contribute as much as it should, and its abnormal appearance may be

recognized, and this tends to localize the abnormality. This lessened contribution often occurs at the upper end of the femur or at the lower end of the tibia. Often, however, the whole extremity is affected, and all the elements contribute to the shortening.

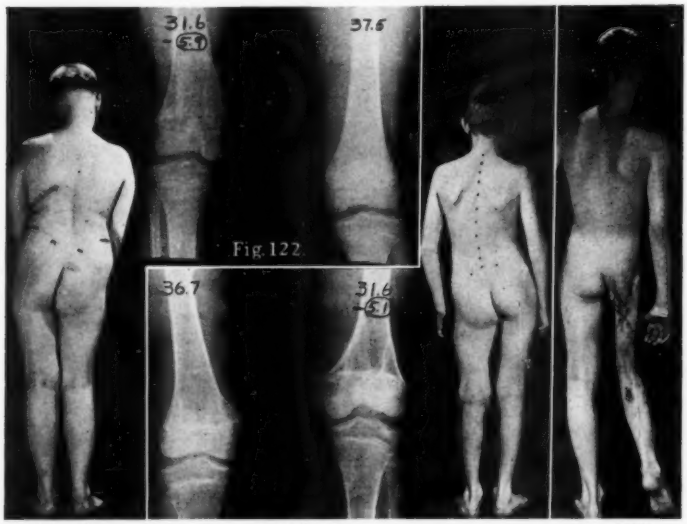


Fig. 122. Fig. 123. Fig. 124.

Fig. 122. Severe inhibition of growth at the right distal femoral epiphysis following osteomyelitis at 4 months of age. The right leg was longer than the left during the 8 months in which drainage continued, but healing was associated with distortion of the bony architecture at the distal epiphysis, and growth on this side was inhibited thereafter. By the age of 10 (above) the right femur was 5.9 cm. shorter and demonstrated 25 per cent inhibition as compared with the normal femur on the left.

Fig. 123. Premature fusion of the left distal femoral epiphysis following fracture involving the lower femoral epiphysis at age 2. By 11 years of age roentgenograms showed closure of the left distal femoral epiphysis with 5.1 cm. of shortening. No growth was contributed by this epiphysis after this age.

Fig. 124. Shortening of the right lower extremity following extensive roentgen therapy for hemangioendothelioma. At the age of 17 months this boy received a total of 2550 roentgen units over the right thigh and leg. Growth was much less rapid on this side, and the discrepancy increased from 2.5 cm. at 4 years of age to about 11.2 cm. at 12 years of age.

Other congenital disturbances or dysplasias which have been classified under the heading of developmental abnormalities may have a marked effect upon growth. Ollier's disease of the diffuse type may cause a severe disturbance in growth (Fig. 121). So too may the more localized type of dyschondroplasia, so-called hereditary multiple cartilaginous exostoses, produce growth disturbances in which for illustrative purposes it might be considered that a part of the epiphysial plate is dis-

placed and is not contributing to the growth of the extremity, but rather to the tumor mass. The effect upon growth of this condition is most dramatically appreciated when one of two parallel bones is affected, as, for example, the radius, whose growth is reduced as the ulna continues to grow, resulting in a bowing deformity of the forearm, a so-called Madelung's deformity.

Injury to the epiphysial plate from one cause or another may occur and may result in one of three states: (*a*) complete stoppage of growth from the plate; (*b*) a reduced increment of growth from the epiphyses; or (*c*) a deformity of growth arising from injury to a certain portion of the plate. Such an injury may arise from infection, as in osteomyelitis, in which the growth cells are actually destroyed. This complication from osteomyelitis is not common in this day of antibiotics, but can and does occur (Fig. 122). Trauma likewise may injure the epiphysial plate with early closure and cessation of growth (Fig. 123). At other times the plate is injured in a particular portion with a resultant deformity and reduced growth. One side of the plate, for example, may close, and the other half may remain open with a rapidly developing deformity as a result. Ordinarily crushing injuries to the plate are the type which interfere with growth, although fractures across the plate, particularly if left unduly displaced, may cause such disturbance. In any fracture affecting the plate, the prognosis must be guarded until the epiphysis has by its performance demonstrated that its growth potential has not been disturbed.

The epiphyses may be injured by other mechanisms, such as roentgen radiation (Fig. 124). The degree of susceptibilty of an epiphysis to radiation depends in part upon the age, with particular susceptibility present in early infancy. Retardation of growth at this age has been described after a soft tissue dose of as little as 800 roentgens to an epiphysis of a child of five months. Such an exposure in an older child would not produce this effect. Exposure of large dosage to an epiphysis must be weighed carefully as to its primary value on the one hand and the possible secondary effects upon growth on the other.

DISCREPANCY IN POLIOMYELITIS

The discrepancy arising from poliomyelitis is the most common cause of significant unequal length of the extremities and warrants particular consideration. It is ordinarily most marked when the disease occurs at a very young age and produces severe paralysis on one side with a normal extremity on the other (Figs. 126, A, and 127, A). After poliomyelitis has affected an extremity a certain amount of inhibition of growth arises on the affected side, and, as the child grows, the extremity becomes relatively shorter and shorter as compared to its fellow. The

effect, however, is not as simple as a certain fixed percentage of inhibition which arises as a result of a certain amount of paralysis. Occasionally, little inhibition of growth occurs with severe paralysis, although as a rule the amount of inhibition roughly parallels the degree of paralysis. The inhibition, furthermore, is far from a straight-line phenomenon. In fact, during the first six months to one year after the disease, slight stimulation of growth rather than inhibition is more likely to occur. After the first year, however, the increment of inhibition ordinarily increases rapidly, so that there is a high degree of inhibition from the second through the fifth year after the onset. After this there ordinarily is a decreasing yearly increment of inhibition.

METHODS OF CORRECTION

Many methods of correcting discrepancies in length in the lower extremities have been proposed and used. Direct leg lengthening and leg shortening have been practiced and in the adult are the only procedures available. Leg lengthening as first described by Codivilla[4] and developed by Abbott[1] was in considerable vogue for many years. The tibia was the chosen site of lengthening in most instances, but it was a formidable procedure and was fraught with complications. An increase of 5 cm. in length could be obtained in a well chosen case. Lengthening is rarely performed now, although it may still be used for particular problems. Shortening is easier to accomplish and is less likely to have complicating difficulties, but is still a formidable operation. It is ordinarily practiced on the femur. Skeletal fixation is essential after the operation, and the tissues readapt themselves satisfactorily to 1½ to 2 inches of shortening. Both lengthening and shortening are formidable operative procedures that require long hospitalization and have certain incidences of disturbing complications. Shortening, furthermore, affects the longer bone, which ordinarily is the normal bone, although the procedure itself is less formidable than is that of lengthening.

In the child the possibility of modifying growth either by stimulation or by inhibition arises. Stimulation would be particularly desirable, since often the abnormal extremity is the shorter one, and thereby with stimulation one might restore the normal contours to the extremity and maintain the height of the patient. Various methods of stimulating growth have been proposed. Sympathectomy to increase the circulation was advocated by Harris and MacDonald,[10] who reported favorable results, as did Barr.[2] Our experiences with sympathectomy have been less favorable, and we do not advocate its use unless it is indicated to improve the circulation of the extremity for reasons other than possible stimulation of growth. The use of dissimilar metals or other irritating substances near the metaphysis has been advocated by Pease[12] and

others. Ferguson[5] suggests the curettage of the medullary canal adjacent to the metaphysis to stimulate growth, and Trueta[15] has proposed the blocking of the medullary canal for the same purpose. The creation of a surgical arteriovenous communication to stimulate growth has also been proposed.[11] The practical value of these techniques designed to produce stimulation has yet to be proved.

Inhibition of growth as a method of correction, on the other hand, has proved to be practical over a period of years. In 1933 Phemister[13] described a method of surgical stoppage of growth by operation on a particular epiphysial plate. In this procedure bone grafts are inserted across the epiphysial plate along with curettage of the plate. Ordinarily the epiphysial arrest of epiphysiodesis is performed on either the lower femoral epiphysis or the upper tibial and fibular epiphyses. This has proved to be a feasible method of inhibiting growth without a disturbing incidence of deformities or complications. In 1949 Blount[3] described the use of staples to straddle the plate as a method of inhibiting growth. This too has proved to be a practical way of stopping growth with a moderate incidence of complicating factors.

If discrepancy is to be corrected by stopping the growth of an epiphysis so as to allow the shorter side to catch up, that is, by epiphysiodesis, it is necessary to be able to predict the correction that would occur from such a procedure. It is essential, therefore, to be able to estimate within reasonable limits the growth of an extremity from any particular age to maturity and to be able to assign the particular portion of growth which is contributed by each epiphysis. A chart has been developed for this purpose which acts as a guide to such predictions with the understanding that actual prediction in a given child is an individual consideration.[6, 7] Skeletal age rather than chronologic age is used with this chart in order to stabilize as much as possible the variable arising from the relative maturity of the patient.

The age at which maturity is attained and thus the number of years of growth remaining for an individual child enters strongly into prediction (Fig. 125). The best practical yardstick available for judging relative maturity is so-called skeletal age as assayed by the use of the Todd[14] or Greulich-Pyle[9] atlas of the skeletal development of the hand and wrist. Predictions of the amount of growth to be inhibited by epiphysial arrest should always be made using skeletal rather than chronologic ages.

Recently we have evaluated the accuracy of prediction in 125 epiphysiodeses in which the patient had reached full growth after having the procedure performed.[8] The actual amount of correction obtained was found to fall within ½ inch of prediction in about 89 per cent.

Certain authors have emphasized that staples may be used without

consideration for prediction of growth with the idea that growth will resume when they are removed if such becomes necessary. It is true that growth may resume after the staples have been removed, but whether this will occur consistently without exception and for how

A B

Fig. 125. Roentgenograms of the hands and knees of 2 girls, each 10 years old, with widely different rates of maturity. *A*, Film of girl who was markedly retarded; her skeletal age was estimated as being about 7½ years, and growth continued in her long bones until the age of 17. *B*, Roentgenograms indicating that the other girl was nearing the end of her growth, the bones being commensurate with those of a 13 year old girl.

long a period a staple may be left in and removed with the assumption that growth will resume cannot be stated. The evidence is that growth may ordinarily be expected to resume after two years of stapling, but if stapling is used at young ages, it is essential that the resumption of growth be dependable without exception. It is our practice, therefore,

to use stapling as an alternate method of arrest, using the prediction table as a guide to its timing.

Epiphysial arrest, both by epiphysiodesis and by stapling, has been proved to be of great value in correcting discrepancy. Over 250 such procedures have been followed in detail in the Growth Study at The Children's Medical Center from the time of operation until each patient has completed his growth. The final results, which have recently been reported in detail elsewhere, were good to excellent. There was a small incidence of deformity arising from both these procedures, so that it was necessary in some instances to perform a secondary operation in order to obtain the desired results, but there were only two patients in our series in whom the results might be said to be totally unsatisfactory. The difficulties that occurred most commonly arose from technical factors in the procedure, such as from asymmetrical fusion of the epiphysis or from a fusion which was either not effective or was slowly effective.

There were only six patients who had an amount of real or potential overcorrection that warranted clinical consideration. Five of these six had femoral epiphysiodeses on the formerly short side in order to prevent overcorrection. One patient ended up with a ½ inch overcorrection and, since he wore a brace on the other side, this was an undesirable amount.

Stapling is not as immediately effective in arresting growth as epiphysiodesis, but was relatively prompt. There was a greater incidence of complications following stapling than after epiphysiodesis, but they were not of great moment.

In general, results in both epiphysiodeses and staplings were most satisfying, and the incidence of complications was insignificant compared with the result attained in almost all cases, namely, an effective predictable correction of deformity.

THE CLINICAL PROBLEM

In considering the clinical importance of shortening in the particular growing child one must consider not only the degree of shortening present at the particular age, but also the potential discrepancy at maturity. A difference of 1 cm. in a boy of 10 years may not be important, but if it is the first evidence of a premature closure of a lower femoral epiphysis, it may well presage a discrepancy of 7 cm. by maturity unless appropriate measures are taken.

A fixed discrepancy of 1 cm. or less is ordinarily not of clinical importance, although even this amount does produce changes in the mechanics of the spine and may, by provoking shearing force, cause some difficulty in adult years by increasing the wear and stress on the

back. A small lift to the heel making up part of the discrepancy may be indicated. Discrepancies of 2 cm. or more ordinarily should be of clinical concern. There may be reasons for making up part of the distance by a lift to the shoe without further procedure. Such a difference, however, may warrant a surgical procedure, provided the patient is of an age for which a simple type of operation is available. The importance of making up the correction is related to factors other than the measurable discrepancy itself. For example, if a shortened extremity by its shortening increases the scoliosis, it is much more important that the discrepancy be corrected. Some patients, by shifting their pelvis and weight, adjust very well with considerable discrepancy when others are obviously thrown off balance and are in difficulty by a similar amount. It is more important to correct the discrepancy in the latter type of patient. A patient who is going to wear a long brace on the short extremity the rest of his life ordinarily is better off with a slight shortening of, say, a centimeter on the affected side, since this allows him to swing the brace forward with easier clearance. The greater the discrepancy, the more is the need to correct it at least in part.

In weighing the need in the individual child for a surgical procedure to correct discrepancy and in considering the choice of procedure it is highly desirable to have observed the growth of the child for a period and indeed to have serial records of growth of the extremities so that the individual pattern of discrepancy of bone growth and bone age is available. One can thus attain a more accurate concept of this problem as it will affect the child at maturity and can more accurately prognosticate both growth and the result of any operation that is contemplated. If the problem is of consequence, observations at at least six month intervals are desirable, including roentgenographic measurements of the lower extremities and films visualizing the epiphyses and wrist films for skeletal maturity. Such measurements are most helpful in evaluating the discrepancy and the factor of inhibited growth, but when it comes time to correct the discrepancy, the amount of lift under the foot required to balance the spine is a determining factor in deciding on the amount of correction that is needed.

In general, in the growing child epiphysial arrests by one technique or another are to be preferred as a method of correction if the amount of discrepancy is of reasonable degree. The arrest is a much less formidable procedure than either lengthening or shortening, requires brief hospitalization, and confines the patient for only a short period. Furthermore, in the result it gives a more normal contour to the extremity than either shortening or lengthening. Arrest allows correction of large discrepancies in a dependable way, whereas methods designed for stimulation provide small amounts at best.

If the shortening is extreme, arrest may not be able to correct the

discrepancy, or indeed it may not be desirable to shorten the extremity to such an extent because of the deforming shortness. If the difference is marked, it may be that a lengthening of the short side may be chosen along with an arrest on the other side. If the short extremity is a distorted one and there are many inches of discrepancy, it may be desirable to adapt the extremity to a prosthesis or to perform an amputation with the fitting of an artificial extremity. Ordinarily discrepancy in the area of 4 or 5 inches may be approached by arrest unless the patient is unusually short. More than this is always open to question. Lengthening, plus arrest, may be indicated for the larger differences. Of course one can leave the short extremity a little short after the corrective procedure and make up further difference in the shoe.

If an arrest is chosen either by epiphysiodesis or by stapling, the timing of the procedure is the important consideration. One must perform the arrest of the particular epiphysis or epiphyses at the time when the particular increment of growth so destroyed will be the amount of correction desired. In practice the use of the prediction chart as a guide to the timing of procedures has worked out satisfactorily, provided it is recognized that it is a guide in making a clinical decision which weighs all the factors involved. Certain difficulties do arise in prediction in a small percentage of cases.[7, 8] In most instances this arises from a shifting pattern of skeletal maturity as judged from films of the wrist.

A child who is interpreted as having a particular skeletal age at the time of an arrest may for some reason shift so that his relative skeletal age changes in relation to his chronologic age. In this way he will either grow for a longer or a shorter period than was originally predicted, depending upon the direction of the shift in relative maturity. In such instances the prediction will go awry. In general, predictions can be made more accurately if the child has been observed over a period of time, both as to the growth of his extremities and the pattern of skeletal age; and if all factors are weighed in making the estimate, a reasonable accuracy in prediction results.

When a large correction is to be attained and both the femoral and upper tibial and fibular epiphyses are to be used, the yearly correction is rapid, and accuracy of prediction is most important. Under such circumstances an epiphysiodesis of the tibia and fibula and a stapling of the femur may be the proper combination. Stapling allows the possibility of resumption of growth when the staples are removed, so that if an error in prediction arises and more correction is obtained than needed, the staples can then be extracted with a reasonable expectation that growth will resume. Often it may be desirable to do an epiphysiodesis at one level, say the tibia and fibula, on one date, observe for

a year or so, and then add the femoral arrest later if it is needed. This diminishes the difficulty arising from errors in prediction.

In doing an arrest for correction it must be remembered that the growth of the shorter side is the factor that corrects a discrepancy. If, for example, a femoral plate on the short side is not growing and is closed, no correction will occur from performing a femoral arrest on the long side. It will prevent further discrepancy, but it will not correct. Arrest of the upper tibia and fibula in addition to the femoral arrest in this case would add the correcting factor. Slowness of growth on the short side must be considered in evaluating the timing of an epiphysial arrest and in estimating the correction that may be expected from it.

In deciding which bone is to be the subject of arrest, when only moderate correction is required, various factors are weighed. Consideration is given as to whether one bone on the short side shows a greater discrepancy than the other. If there is a marked difference, the level of the knee is better preserved by performing the arrest on the bone corresponding to the short bone on the other side. Every consideration should be given to the fact that in girls particularly, length of the tibia is important on an esthetic basis, and often a procedure on the femur may be preferred for this reason. Stapling of a tibia and fibula is not as desirable a procedure as stapling of the femur and is accompanied by a greater likelihood of complicating difficulties.

After any operative procedure affecting growth it is important to observe the patient at regular intervals, using roentgenographic records as well to make sure that the desired effect is obtained. One is then in a position to take measures at that time which will avoid an untoward result.

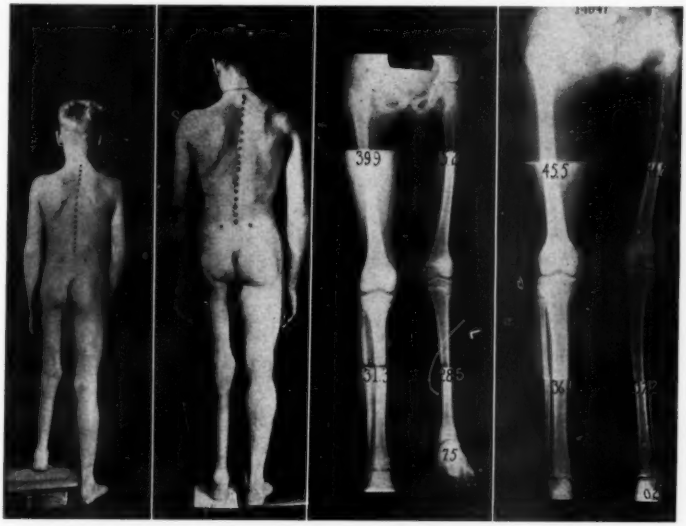
CASE REPORTS

The factors involved in the correction of discrepancy by epiphysial arrest may be illustrated by considering individual case reports.

CASE I. A boy had poliomyelitis at the age of 3 years with extensive paralysis of the left lower extremity and little or no residual paralysis elsewhere (Fig. 126). There was progressive discrepancy in the length of his lower extremities, so that 7½ years after the onset his bone discrepancy as measured by orthoroentgenograms was 7.5 cm. Since the height of his foot had been reduced by a Hoke arthrodesis and countersinking at the age of 7½ years, it required a block 9 cm. in height under his foot in the standing position to balance his pelvis and spine.

When he was 10 years and 10 months of age and at a time when his estimated skeletal age was 10½ years, an epiphysiodesis of the upper tibial and fibular epiphyses was performed with the idea that a femoral arrest would be performed at a later date as indicated. At the time of this first arrest it was predicted that this procedure would correct the discrepancy by 4.3 cm., based upon the median of the prediction chart. By the same prediction it was considered that a femoral arrest would be indicated at a skeletal age of 12 years, which, if it maintained the same relation to chrono-

logic age, would be when he was 12 years and 4 months old. In fact, however, his bone age became accelerated, so that a year later when he was only 11 years and 10 months of age his bone age was already 12½ years, and a femoral arrest was performed at this time. The median predicted effect from this procedure at this age was 4 cm. In fact, he obtained almost exactly 4 cm. from the femoral arrest and 3.9 cm. from the tibiofibular arrest. Orthoroentgenograms taken before the procedure showed the total difference in bone length as 7.5 cm., and final bone length in orthoroentgenograms taken at 15 years and 3 months when he had a skeletal age of 17 years and 3 months showed a remaining discrepancy of only 0.2 cm. He still required about 1.2 cm.



A B C D

Fig. 126. A and B, A total discrepancy of 3½ inches (7.5 cm. in bone lengths) resulted in this 11 year old boy from paralysis of the left lower extremity due to poliomyelitis at the age of 3. C and D, At maturity the same boy had leg lengths that were essentially equal. Epiphysiodeses of both the right lower femur and upper tibia and fibula had controlled and corrected the increasing discrepancy.

under his heel to completely balance his pelvis, but since he wears a brace part-time this was a desirable discrepancy. Potential discrepancy without surgery was estimated to have been 11.8 cm.

This boy required a large amount of correction, and he illustrates the principle of performing an arrest on one epiphysis early and then adding the other epiphysis at a later date so as to minimize errors in prediction. He illustrates, too, how skeletal age may shift in relation to chronologic age, but beyond everything else he demonstrates how relatively simple procedures performed at the proper time can reduce deformity and improve function. He no longer needs to wear a heavy raised sole and can control his extremity with the limited musculature on this side.

CASE II. A girl had the onset of acute anterior poliomyelitis at the age of 2½ years with severe involvement. The left lower extremity remained essentially flail, but the right showed good recovery.

Marked inhibition of growth was noted in the left leg, beginning about 18 months after onset, and by 6½ years after the initial disease the left leg was already 6.1 cm. shorter than the normal right side. When she was 9 years and 5 months of age and had a skeletal age of 10 years and 3 months, an epiphysial stapling was performed. The median of predicted effect at this age was 4.5 cm., and in reality she obtained

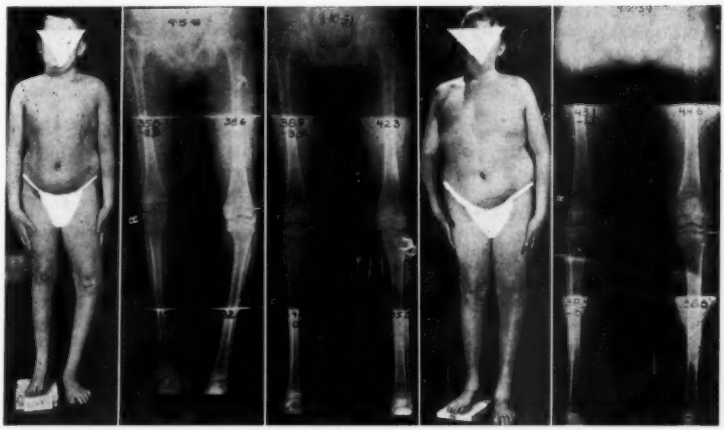
A B C

Fig. 127. A and B, Shortening of the left lower extremity 6 years after paralysis of the left leg following poliomyelitis. Retardation of growth on the left amounted to 25 per cent as compared to the normal side, and the total discrepancy was already 6.1 cm. at the age of 9 years. C, Orthoroentgenogram of the same girl at the completion of her growth (skeletal age 15¾ years) shows equal leg lengths following stapling of the right femur and epiphysiodesis of the right tibia and fibula which had sufficiently slowed down the growth of the right side to allow the left to make up the discrepancy. The left femur is now 2.1 cm. longer and the left tibia 2.3 cm. shorter than on the right.

4.6 cm. The femoral arrest was chosen first in order to develop most of the shortness in this bone, since she was female, and it was proposed to maintain her tibia at a reasonable length for appearance's sake. In order to obtain enough correction an epiphysial arrest by epiphysiodesis of the upper tibial and fibular epiphyses was performed 14 months later when she had a skeletal age of 12 years and 3 months, and from this she obtained an additional 1.9 cm. The final result is that she is within 2 mm. of equal length in the long bones of the two sides (Fig. 127). She has some slight shortness remaining from the reduced height of her foot, but this is very desirable since she must wear a brace on this side permanently. She no longer requires any lift on her shoes, and her gait is very much better with the lighter equipment. The

correction of her discrepancy in this flail leg which requires a brace permanently is of less importance, however, than if the leg had enough musculature and control that it might go without a brace.

This young lady illustrates the use of the combination of stapling and arrest when a large amount of correction is required and in particular when some difficulty arises in prediction due to either a skeletal



A B C D E

Fig. 128. *A* and *B*, Photograph and orthoroentgenogram showing 4.3 cm. shortening of the right lower extremity following severe coxa plana and a marked genu varum of the left upper tibia. *C*, Same case 2 years after staples had been inserted in the lateral aspect of the left upper tibia and fibula to correct the varus. *D* and *E*, Photograph and orthoroentgenogram to show the corrected contour of the left leg 3 years after the staples had been removed. Femoral shortening is being corrected by epiphysiodesis of the right lower femur. The boy was still growing at the age of 17 (skeletal age, 15).

age that is difficult to interpret or, as in this instance, to an irregular factor of inhibition.

The tibiofibular arrest was by epiphysiodesis, which is a definitive procedure that stops growth promptly and is unlikely to require further intervention. The choice of stapling for the femur gave a leeway if the prediction had turned out to be inaccurate and overcorrection had been threatened. The staples could then have been removed with the idea that growth would resume from this plate. Often the tibiofibular arrest is performed first with the femur added later, or the two levels may be performed at the same time. In this instance the arrest of the femur was done first in order to get most of the correction from this bone rather than produce an undesirably short, homely lower leg. As it turned out, the prediction was accurate, and it was not necessary to remove the staples.

CASE III. A boy at the age of 6 years first had symptoms of coxa plana on the right side. This went untreated for 2½ years, and after this he was treated in a nonweight-bearing splint for 3½ years until the head of the femur had healed. He had, in addition, an osteochondrosis deformans tibiae on the left side. At the age of 12 years his right leg was 4.3 cm. shorter than the left, and there was a 23 per cent inhibition on this side compared with its fellow. The tibial deformity on the left was marked (Fig. 128), and stapling was performed on the lateral side of the tibial epiphysis and of the fibular epiphysis for this varus deformity of the tibia. The bone was straight 2 years later and the staples were removed. After this both sides of his left tibia have grown equally as demonstrated by the even transverse lines present in the roentgenograms of the tibia taken 3 years after the tibial staples were removed. The femoral shortening is being corrected by an epiphysiodesis of the lower femoral epiphysis on the left. This was performed when the boy was 15 years of age and his skeletal age was 13 years. Two years later the discrepancy of the femoral lengths had been reduced to 1.5 cm. The boy is still growing, and correction is in process.

This case illustrates two features, one, the use of staples to correct epiphysial deformities of this severe type which will not correct themselves, and that equal growth may resume after the staples are removed. The value of an arrest in correcting the discrepancy in length is well demonstrated.

As may be seen in each of these cases, the problems of growth are individual to each child. Judgments regarding treatment involved, first, a careful analysis of the cause of the discrepancy and its implications at maturity and, secondly, a soundly established decision as to whether surgical intervention is indicated and, if so, as to the type that should be performed. In any instance, early recognition of the discrepancy and regular observation as growth occurs permit the best possible solution to the problem. In particular, the value of epiphysial arrest as a method of correcting discrepancy must be appreciated both as to its effectiveness and the need for performing it at exactly the right age to obtain the desired correction.

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